Attachment I to Vinyl Institute Comments on Center for Biological Diversity Petition

The purpose of this attachment is to list studies, reviews and other information cited in the Petition and demonstrate that they are irrelevant or unsupportive of the Petition's central claims regarding the potential hazard of discarded PVC products.

The columns contain the Petition page number with cited reference, the Petition's characterization of the reference, Reference Language excerpts, and Vinyl Institute observations, if any.

- Including studies in this matrix does not represent agreement that the studies were properly conducted or relevant to discarded PVC products.
- The study author's conclusions are not critiqued, only how much the reference supports the Petition's claims in this context.
- The Vinyl Institute's observations here do not replace our narrative comments.
- As explained in the Vinyl Institute comments, most of the referenced studies are irrelevant given current regulations, legislative and statutory developments since 2014, and the absence of the risk inherent in discarded PVC products.
- This matrix does not address legal or regulatory arguments raised in the Petition.

Attachment I: Vinyl Institute Comments on Center for Biological Diversity Petition References

No.	Page & Reference	Petition Characterization	Reference Language	VI Observations
			Introduction	
1.	Intro., p. 1 FN 5 – Cózar (2014)	Scientists, scholars and concerned citizens have long warned that inadequate waste management strategies are contributing to the widespread degradation of the marine environment.	*Parenthetical:* (reporting that "the intense consumption and rapid disposal of plastic products" has contributed to the accumulation of "tens of thousands of tons" of plastic debris in surface waters of the open ocean) "The plastic concentrations per surface area were comparable across each of the five accumulation zones, although the North Pacific Ocean contributed importantly to th global plastic load (between 33 and 35%), mainly owing to the size of this gyre. The plastic load in the North Pacific Ocean coul be related to the high human population on the eastern coast of the Asian continent, the most densely populated coast in the world, with one-third of the global coastal population (20)."	No direct link to U.S. mismanagement. While the study acknowledges marine plastic pollution, it does not mention PVC specifically or any other specific polymer.
2.	Intro., p. 1 FN 5 – Barnes (2009)		*Parenthetical:* (explaining that "plastic persists in landfill sites and if not properly	CBD's reliance on this article also is beyond general views on plastic waste. The paragraph reproduced here and a table

			buried may later surface to become 'debris'") "Municipal waste is dominated by containers (e.g., drink bottles) and films (e.g., carrier bags, packaging sheets), agricultural waste may contain large quantities of a single film and C&D waste may contain polyvinyl chloride (PVC) pipe and large plastic containers."	are the only mentions of PVC in the article. The article states: "Mega-debris at sea was highlighted by tens of thousands of each of basketball shoes, hockey gloves and bath toys released from containers washed off of ships (Weiss et al. 2006). . . . Typically, 40–80% of mega- and macro-marine debris items are plastic, much of it packaging, carrier bags, footwear, cigarette lighters and other domestic items (Derraik 2002; Barnes 2005)." "The most common items are plastic films, such as carrier bags, which are easily wind blown, as well as discarded fishing equipment and food and beverage packaging." It also acknowledges that "[t]he major release of plastics to the environment is the result of inappropriate waste management and improper human behaviour, e.g. littering (abandoning waste away from collection points)."
3.	Intro., p. 1 FN 6 – Barnes (2009)	As much as eighty percent of ocean litter consists of lightweight and durable plastic trash, which poses a range of serious threats to aquatic organisms and human beings.	Citing to same page as above.	No link to PVC.
4.	Intro., p. 1 FN 7 – Galgani (2013)	A recent review of the scientific literature revealed that nearly four hundred species have ingested or become entangled in marine debris, representing an increase of more than forty percent over the previous survey, published only sixteen years before.	Over half of the reported species (about 370) were associated with entanglement in and ingestion of marine debris, representing an increase of more than 40% since the last review in 1997, when 247 species were reported affected by those two impact categories (Laist, 1997).	No reference to PVC. The article notes regarding marine litter: "[i]n terms of plastic litter-type or use, in year 2012, rope and netting accounted for 57% of encounters, followed by fragments (11%), packaging (10%), other fishing-related litter (8%) and microplastics (6%) (CBD, 2012)." Regarding fragments, the article cites Barnes and states: "[a]n emerging area of concern is the accumulation of microplastic fragments in the water column and in sediments (Thompson et al., 2004). Pieces of common polymers (including polyester, nylon, polyethylene and polypropylene) of less than 20 mm have been recorded in the marine environment worldwide."
5.	Intro., p. 1 FN 9 – Browne (2010)	Improperly discarded PVC constitutes a substantial proportion of ocean litter and poses especially	In contrast to microplastic debris, assemblages of smaller microplastic fragments were mainly composed of denser plastics such as polyvinylchloride (26%), polyester (35%), and polyamide (18%).	This study was limited to the Tamar estuary in the U.K. While finding that microplastics comprise up to 65% of the total debris found, PVC was only 26%. Note that percentages are based on 952 debris items in 30 samples of sediment. No relationship to U.S. waste management.

		significant threats to human and environmental health		
6.	Intro., p. 1 FN 10 – Mulder (2001)	Even before the production of this material began to accelerate in the 1930s, researchers suspected that PVC's primary building block, vinyl chloride, produced toxic effects in laboratory animals.	Even before the large-scale production of the 1930s, PVC's primary building block vinyl chloride (VC), was thought to be a potentially toxic substance, causing fatty degeneration in the liver and kidneys of test animals. In 1938, the first evidence of these toxic effects in animals was published. By the end of the 1950s, the toxic effects were even clearer. VC could be linked to acro-osteolysis (characterized by clubbed fingers, bone deterioration, heart and metabolic problems, skin changes, and muscle anomalies), and Raynaud's Syndrome (highly sensitive, cold and prickling fingers). In the 1960s, an increasing number of reports in other countries described VC factory workers (especially those occupied with cleaning reactor vessels) who displayed these diseases and other symptoms. Many reports stemmed from the industry itself. For example, Dow Chemical and B.F. Goodrich carried out studies of their employees. In Germany, by September 1974, 167 suspected cases of 'VC disease' had been reported.	The toxicity of vinyl chloride monomer is well-studied and resulted in strict regulations, which, in turn, drove changes in production technology. The study does not inform the management of discarded PVC products.
7.	Intro, p. 2 FN 12 – Sass (2005)	experts continue to argue that the agency's risk assessments are inadequate	*Parenthetical:* (finding that EPA's assessment of vinyl chloride "downplay[s] risk" and reflects excessive industry participation)	As stated in the 2014 VI comments response, this paper (Sass) was criticized in a letter to the editor of the publishing journal as "a case study of misrepresentation," for not "includ[ing] or address[ing] recent studies characterizing the weight of the scientific evidence related to vinyl chloride and [making] inaccurate and unsupported allegations about the integrity of the U.S. Environmental Protection Agency (EPA) scientists and the rigorous peer review process utilized by the U.S. EPA."

8.	Intro., p. 2 FN 12 – Kielhorn (2000)		*Parenthetical:* (explaining that vinyl chloride "remains a cause for concern because potential exposure to this chemical and new cases of [related cancers] are still being reported") But recent epidemiological, environmental, and biomechanistic findings have opened up new aspects of this chemical. VC has recently been evaluated by an international interdisciplinary task group; this paper highlights these recent developments, which are presented in more detail in the International Programme on Chemical Safety (IPCS) Environmental Health Criteria document on VC. VC remains a cause for concern because potential exposure to this chemical and new cases of ASL [angiosarcoma of the liver] are still being reported.	Mundt KA, Dell LD, Crawford L, et al., "Quantitative estimated exposure to vinyl chloride and risk of angiosarcoma of the liver and hepatocellular cancer in the US industry-wide vinyl chloride cohort: mortality update through 2013", Occup Environ Med 2017;74:709–716 provides the most recent study of ASL cases in the U.S. According to this study: "No clear exposure–response relationships were observed for mortality from other cancers, including lung cancer, brain cancer, non-Hodgkin's lymphoma or melanoma. And Risks of ASL and HCC were only elevated among workers with very high estimated cumulative exposures, that is, over 1000 parts per million-years, and after long latencies (median latency was 36 and 48 years, respectively, for ASL and HCC)." The total cohort of worker history studied were 9,951 men employed in the vinyl chloride industry covering health records from 1942 to 2013. Of this cohort, 63 cases of ASL, 32 cases of HCC and 36 unspecified cases of liver cancer mortality were confirmed."
9.	Intro., p. 2 FN 13 – Stern (2008)	In addition to vinyl chloride, PVC contains significant concentrations of regulated and unregulated chemical additives, including phthalate plasticizers and heat stabilizers mixed from lead, calcium, barium and cadmium.	In discussing polymeric tubing materials and additives used for drinking distribution in homes and buildings, the article states: "Heat stabilizers have included metal mixtures of lead (Pb), calcium (Ca), barium (Ba) and/or cadmium (Cd) (Al Amack et al. 2000). Organotins currently dominate the U.S. market for this purpose (Tullo 2000)."	As noted in the 2014 and current comments, lead and cadmium have essentially been eliminated from PVC products (though may still be present in imported products or specialty applications). Specifically, lead stabilizer use was discontinued from wire and cable applications in 2006, the last known use of lead in the U.S. PVC industry. PVC pipe never used lead stabilizer. PVC profile extrusions substituted tin stabilizers for lead beginning in the late 1960's and had transitioned completely away from lead stabilizer by the early 1990's.
10.	Intro., p. 2 FN 14 – Stern (2008)	Recent studies reveal that finished PVC products leach significant concentrations of these compounds into the environment as they	There are numerous chemical substances that can be released from polymeric materials into drinking water over time. When plastic pipes are used for residential plumbing and/or in the distribution humans may be exposed to a variety of chemical	According to the study acknowledgement, this study was partially funded by the International Copper Association. As the study notes, very little data on leachates from PVC and CPVC pipe is available because the pipe and fitting materials are tested and certified to meet NSF Standard 61, which was

		deteriorate with age, threatening severe biological consequences.	leachates . . . Based on available data, this review has shown that numerous chemical substances in polymeric tubing have the potential to migrate from the PV into drinking water. Migration may vary significantly among pipes, depending on the materials/resins used in manufacturing and extrusion, differing location/environmental conditions and usage patterns. The possible health effects of some leachates have been studied in animals and/or humans. The majority of constituents or additives in plastic pipes have not been monitored and/or evaluated toxicologically.	developed in 1976 and adopted by EPA in 1986 for assuring the health and safety of drinking water components. NSF 61 testing assures that PVC components will not leach vinyl chloride over the life of the pipe (100 years) at levels that would result in a drinking water concentration above EPA's maximum contaminant level (MCL) limit of 2.0 ug/L. According to data collected pursuant to EPA's Six Year Review of Drinking Water Contaminants (*see* ASCII delimited file *SYR3_PhaseChem_4.zip (o-Dichlorobenzene to Xylenes)*, available from EPA at: [HYPERLINK "https://www.epa.gov/dwsixyearreview/six-year-review-3-compliance-monitoring-data-2006-2011"]) only 125 readings out of 370,000 samples analyzed were above the MCL. In other words, detections of VC are practically nil (0.033%) in this data set. Another study funded by the American Water Works Association Water Research Foundation. Walters, R., et al., "Investigation of factors affecting the accumulation of vinyl chloride in polyvinyl chloride piping used in drinking water distribution systems", Water Research 45 (2011) 2607-2615, Feb. 19, 2011, examined PVC and CPVC pipe for vinyl chloride leachate. The study did detect vinyl chloride (300 ng/L after 2 years) but at levels below EPA's MCL of 2.0 ug/L. Interestingly, the study also determined that vinyl chloride is detectable in copper pipe systems as a secondary disinfectant byproduct, but again below EPA's MCL: "Preliminary evidence suggests that VC may accumulate not only via chemical leaching from the plastic piping, but also as a disinfection byproduct (DBP) via a chlorine-dependent reaction. This is supported from studies with CPVC pipe reactors where chlorinated reactors accumulated more VC than dechlorinated reactors, copper pipe reactors that accumulated VC in chlorinated reactors and not in dechlorinated reactors, and field samples where VC levels were the same before and after flushing the lines where PVC/CPVC fittings were contributing. Free chlorine residual tests suggest that VC may be formed as a secondary, rather than primary, DBP."

11.	Intro., p. 2 FN 15 – Swan (2005)	For example, one-quarter of U.S. women already exhibit concentrations of phthalate metabolites higher than those correlated with irregular sexual development in male infants,	The median concentrations of phthalate metabolites that are associated with short AGI and incomplete testicular descent are below those found in one-quarter of the female population of the United States, based on a nationwide sample. These data support the hypothesis that prenatal phthalate exposure at environmental levels can adversely affect male reproductive development in humans.	Phthalates are not present in most PVC products, and the relationship to discarded PVC products is not addressed in this reference.
12.	Intro., p. 2 FN 16 – Adibi (2003)	and scientific evidence indicates that contamination might be even more prevalent in urban settings.	*Parenthetical:* (reporting that pregnant women in New York City "appear to be exposed [to phthalates] at levels above background levels in the United States, which may have implications for their pregnancy and/or the fetus") Nonetheless, the evidence indicates that pregnant women in New York and Krakow are experiencing a range of exposure levels to phthalates with some extreme values that may be associated with a biologic response. The New York women appear to be exposed at levels above background levels in the United States, which may have implications for their pregnancy and/or the fetus. These results require further investigation. A molecular epidemiologic study is being carried out to more thoroughly characterize exposures in these two cohorts and to incorporate placental markers of in utero endocrine disruption that may be related to placental function and pregnancy outcomes.	The article mentions the use of DEHP as the 'primary plasticizer' in PVC since the 1930s while introducing the various uses of phthalates in everyday items. As our main comments note, plasticizers and their use has shifted.
13.	Intro., p. 2 FN 17 – Bidoki (2010)	Despite its status as "one of the most hazardous consumer products ever created,"	Because of all these risks and hazards, PVC plastic, commonly referred to as vinyl, is known as one of the most hazardous consumer products ever created. PVC is believed to be dangerous to human health and the environment throughout its entire life cycle, at the factory, in our homes, and in the trash.	CBD mischaracterized this reference, as the article goes on to say: "Recent developments in introduction of non-toxic plasticizers, well planned production plants and strictly observed incineration and disposing procedures are gradually changing people's attitude towards PVC." Indeed, using an eco-efficacy approach, the article concludes "PVC plastisol was found as the most eco-efficient coating agent followed by polyacrylate and polyurethane resin dispersions in water."

14.	Intro., p. 2 FN 18 – Rochman (2013)	PVC and its associated chemical additives are managed in much the same way as food scraps and grass clippings after disposal.	Plastic debris can physically harm wildlife. Moreover, many plastics may be chemically harmful in some contexts — either because they are themselves potentially toxic or because they absorb other pollutants. Yet in the United States, Europe, Australia and Japan, plastics are classified as solid waste — so are treated in the same way as food scraps or grass clippings.	No source is offered to support the statement. Although the article addresses PVC, it does not address disposal beyond this overly broad and unsupported comment. Any material can be harmful to wildlife if not managed properly, but mismanagement is not demonstrated.
15.	Intro., p. 2 FN 19 – Sadat-Shojai (2011)	Americans already discard billions of pounds of this plastic each year, and experts anticipate that annual waste generation will increase significantly in the near future, as durable products and construction goods reach the end of their useful lives.	Moreover, although the long-life PVC products show a long service life and there is a long time-lag between PVC consumption and the amassing of PVC waste, they will finally become waste at a certain point in time [2,8,16,17]. As a result, the quantity of used PVC items entering the waste stream is gradually increased as progressively greater numbers of such PVC products approach to the end of their useful economic lives [1,9,10].	This article focuses on recycling as an alternative to landfilling or incinerating PVC goods that have reached end-of-life. Although the article concludes that none of the recycling approaches – at the time (2011) – are completely satisfactory, it does not foreclose this pathway. Recycling is addressed in our narrative comments.
16.	Intro., p. 2 FN 20 – Depledge (2013)	Marine plastic pollution harms ocean organisms, threatens ecological integrity and damages human health and prosperity	On the 5th and 6th June 2013, ca. 100 scientists from 6 countries met for a workshop at the University of Siena, Italy, to review current knowledge and to clarify what is known, and what remains to be investigated, concerning plastic litter in the sea. The content of the workshop was designed to contribute further to the European Marine Strategy Framework Directive (MSFD) . . . In addition, a number of statements relevant to policymakers were prepared by the organisers at the end of the meeting which are presented here, and which had the overwhelming support of the workshop participants . . . 2) What we know concerning plastic litter in the sea . . . 3) Some kinds of marine organisms are particularly vulnerable to plastic litter, including turtles, marine mammals, suspension feeders and deposit feeders . . . 5) Plastic litter can play a role in facilitating the introduction of invasive species into	This article reports a number of statements from the June 2013 workshop at the University of Siena (Italy) that were relevant to policymakers and scientists and for which there was overwhelming agreement. The workshop was designed to contribute further to the European Marine Strategy Framework Directive (MSDF). The six attending countries were not identified. The article posits that workshop may have been informed by experiences and knowledge of plastic litter management in Europe. The report also does not specifically address PVC or discarded PVC products, but rather marine plastic litter as a general matter.

			new localities, thereby influencing both biodiversity and ecosystem structure and functions in some areas.	
§ I.A. The Resource Conservation Recovery Act – Statutory Background				
I.B.1. Discarded PVC Contains Toxic Constituents				
17.	§ I.B.1., p. 7 FN 65 – Allsop (2012)	Moreover, because PVC is intrinsically unstable, the commercial viability and almost unlimited versatility of this material derive from complex formulations of chemical additives, which frequently include multiple toxic constituents.	Due to its unique combination of properties, PVC is never handled on its own. Instead, a complex formulation incorporating several additives is used. A typical base formulation contains: PVC resin, heat stabilizer(s), internal lubricant(s), external lubricant(s), processing aid, pigment, UV stabilizer, as well as primary and secondary plasticizers . . . PVC is intrinsically unstable because of molecular defects in some of the polymer chains . . . and when subject to heat they initiate a self-accelerating dehydrochlorination reaction. Stabilizers neutralize the HCI produced and introduce nucleophilic substitution reactions that prevent further degradation.	PVC is not "intrinsically unstable." Many PVC products are durable goods with extended estimated useful lives.
18.	§ I.B.1., p. 7 FN 65 – Cadogan (2012)		*Parenthetical:* ("PVC would be of little use" as a commodity polymer "[w]ithout the wide range of additives available.") Over 90% of plasticizer sales by volume are into the PVC industry. The reason is that the benefits imparted by the plasticization of PVC are far greater than those imparted to other polymers. PVC stands alone among polymers in its ability both to accept and retain large concentrations of plasticizer. This is due to a morphological form consisting of highly amorphous, semicrystalline, and highly crystalline regions. Without the wide range of additives available (e.g., plasticizers, stabilizers, fillers, lubricants, pigments) PVC would be of little use. The development of PVC as a commodity polymer is fundamentally linked to the development of its additives.	As stated in our 2014 comments to the Petition, 75-80% of all PVC products are rigid and do not contain plasticizer.

19.	§ I.B.1., p. 7 FN 66 – Stern (2008)	For example, stabilizers mixed from lead, barium and cadmium are often employed to facilitate high-temperature manufacturing processes.	This review evaluates the literature on the occurrence of regulated and unregulated substances in drinking water related to the use of plastic pipes, characterizes potential health hazards, and describes uncertainties associated with human health and exposure in need of further research. Polymeric plastics used for drinking distribution in homes and buildings include polyethylene (PE), high density polyethylene (HDPE), medium density polyethylene (PEM), low density polyethylene (PEL), cross-linked polyethylene (PEX), polyethylene of raised temperature resistance (PE-RT), aluminum cross-linked polyethylene (Al/PEX), polypropylene (PP), unplasticized polyvinyl chloride (PVC), and chlorinated polyvinyl chloride (CPVC). The use of these materials resins alone or in combination in drinking water systems varies widely among developed and developing countries (Trew et al. 1990). There is a trend toward the use of PE in Europe, whereas chlorinated resins are common in the United States (Trew et al. 1990; Brocca et al. 2002). Heat stabilizers have included metal mixtures of lead (Pb), calcium (Ca), barium (Ba) and/or cadmium (Cd) (Al Amack et al. 2000). Organotins currently dominate the U.S. market for this purpose (Tullo 2000).	As noted above and in our comments, lead and cadmium have essentially been eliminated from PVC products, and the referenced stabilizers are not used in PVC pipe.
20.	§ I.B.1., p. 7 FN 67 – Koch (2009)	In addition, the industry relies heavily upon dialkyl- and alkylarylesters of 1,2-benzenedicarboxylic acid, commonly known as phthalate plasticizers, to impart a range of beneficial properties to [a] myriad [of] consumer, construction and industrial goods.	The term phthalates describes a class of chemicals that are dialkyl- or alkylarylesters of 1,2-benzenedicar- boxylic acid. Their industrial applications are related to the length of their ester chain. Phthalates with alkyl- chain lengths from 3 to 10 carbons are widely used as general-purpose plasticizers in polymers, primarily in polyvinyl chloride (PVC) resins. Within soft PVC, the plasticizing phthalate content can be up to 40 percent. Typical products	Not all vinyl materials contain phthalate plasticizers. In fact, 75-80% of PVC products do not contain any plasticizer. These articles address the function of plasticizers and not the management of discarded PVC products.

			containing phthalates are floorings, roofings, wall coverings and cables, clothing, packaging materials and toys (David et al. 2001; EC 2008).	
21.	§ I.B.1., p. 7 FN 67 – Cadogan (2012)		A plasticizer is a substance incorporated into a material to increase its flexibility, workability, or distensibility. A plasticizer may reduce the melt viscosity, lower the temperature of the second-order transition, or lower the elastic modulus of the product.	
22.	§ I.B.1., p. 7 FN 67 – Talsness (2009)		*Parenthetical:* (explaining that "the addition of phthalates makes brittle [PVC] soft") Phthalates function as plasticizers to give flexibility to high-molecular-weight polymers and are found in soft plastic products (the addition of phthalates makes brittle polyvinyl chloride (PVC) soft).	
23.	§ I.B.1., p. 7-8 FN 68 – Rahman (2004)	These compounds may comprise up to eighty percent of finished PVC, depending upon desired characteristics.	The current paper presents a brief history and an overview of the traditional plasticizers currently available in the world market, discusses some of the problems associated with the end uses of these plasticizers and reviews recent scientific approaches to resolve these problems. Phthalate plasticizers have been a target of worldwide scrutiny in the past two decades from consumer and environmental groups on the grounds of potential carcinogenicity and possible endocrine modulating effects [14]. PVC-based medical plastics have received the most attention partly because medical uses constitute 10% of the phthalate plasticizer market [23]. The PVC used in IV and blood storage bags typically contain 30-40% wt DEHP and medical tubing such as dialysis tubing may contain as much as 80% wt DEHP.	As stated in our 2014 comments, this article overstates the presence of phthalates. Medical bags and tubing contain only 80 parts per hundred of resin, or about 45% DEHP. The use of 80% DEHP, as CBD suggests, would not produce a usable product. Even if this level of phthalate plasticizer was used, medical devices such as dialysis tubing are closely evaluated by the Food and Drug Administration for safety and would be subject to disposal as medical waste. In addition, only flexible vinyl products use significant amounts of plasticizers and not all of these plasticizers are phthalate esters – e.g., citrate plasticizers, such as triethyl citrate. In 2012, about 90% of all plasticizer volume went into PVC and about 62% of that amount was the phthalate type.
24.	§ I.B.1., p. 8 FN 68 – Allsop (2012)		*Parenthetical:* ("Each producer makes a range of PVC polymers which vary in	This reference reinforces the fact that PVC is not appropriately characterized as a single product or substance. Indeed, PVC resin recipes vary widely. As such, it is

			morphology and in molecular mass, depending on the intended end use.") Poly(vinyl chloride) is a generic name. Each producer makes a range of PVC polymers that vary in morphology and in molecular mass, depending on the intended end use.	inaccurate to characterize PVC products in a single brush stroke, as CBD has here.
25.	§ I.B.1., p. 8 FN 69 – Kastner (2012)	Despite the availability of less harmful alternatives,	The research presented in this paper aims to quantify and compare the leachability of potential "green" alternative plasticizers to that of DEHP. A method was developed to assess leaching of several poly(vinyl chloride) (PVC) plasticizers in aqueous media using gas chromatography (GC), and compared to a gravimetric standard test method (ASTM Method D1239). The GC method was a more direct measurement of plasticizer concentration in the aqueous phase. Several "green" plasticizer candidates were found to minimize aqueous leaching to rates ten times below that of DEHP; notably dioctyl succinate (DOS), dihexyl maleate (DHM), methyl cyclohexyl diester (MCDE), diethylhexyl succinate (DEHS), hexanediol dibenzoate (C6), and the commercially available Hexamoll® DINCH. The method developed and the trends presented in this paper can be used as a tool in designing a "green" plasticizer with favorable leaching properties. Not only did plasticizers containing a longer hydrophobic hydrocarbon chain or containing a less polar functional group show a drastically reduced leaching rate compared to DEHP, but the DINCH, DOS, DHM, DEHS and MCDE plasticizers showed no leaching at all within the three week time frame of the leaching experiments. In terms of the criterion of	Kastner describes a method for designing a green plasticizer and presents trends on the topic. Although the paper identified several alternative plasticizers that may be viable options due to low leachability – e.g., DINCH, DOS, DHM, DEHS, MCDE, and C6 – only one of these (C6) was noted to be commercially available. This further supports the view that phthalate plasticizers face competition and are not the exclusive means of making vinyl resin into flexible PVC products.

			reduced leachability, these are ideal candidates for a “green” PVC plasticizer. The C6 plasticizer also exhibited a very slow leaching rate, and the fast degradation of its metabolites in the environment shown previously, makes it also worthy of consideration as a replacement to DEHP.	
26.	§ I.B.1., p. 8 FN 70 – Brandt-Rauf (2012)	the industry consumes over fifteen billion pounds of vinyl chloride	Polyvinyl chloride (PVC) is one of the most commonly manufactured plastics in the world, used in a wide variety of products including packaging, pipes, automotive parts, construction materials and furniture. PVC is polymerized from vinyl chloride (VC) monomer, which is one of the highest production volume chemicals globally with a current annual worldwide demand of approximately 16 billion pounds which is increasing at an approximate 3% annual rate. Up to 98% of VC is used in the production of PVC.	
27.	§ I.B.1., p. 8 FN 71 – Chatterjee (2010)	and sixteen billion pounds of phthalate plasticizers each year.	Globally, more than 18 billion pounds of phthalates are used each year primarily as plasticizers in flexible polyvinyl chloride (PVC) products (Blount et al. 2000a) and also as inert ingredients in many sprays including pesticides and in many consumer products such as cosmetics and wood finishes (Blount et al. 2000b).	As discussed in more detail at line 85, the VI estimates 1,130 million pounds of plasticizer was used in PVC in 2019. Of that, about 50% of the plasticizer was a non-phthalate type.
28.	§ I.B.1., p. 8 FN 71 – Lithner (2009)		To make the PVC soft, a high percentage (10–50wt%) of plasticisers is added (Andrady, 2003). Over 98% of all plasticisers are used in PVC, and 95% of all plasticisers are phthalates (OECD, 2004).	
29.	§ I.B.1., p. 8 FN 75 – Heudorf (2007)	These substances are not chemically bound to PVC and, thus, enter the environment naturally as discarded plastics deteriorate with age, resulting in pervasive contamination.	This paper presents an overview on current risk assessments done by expert panels as well as on exposure assessment data, based on ambient and on current human biomonitoring results. As the phthalate plasticizers are not chemically bound to PVC, they can leach, migrate or evaporate into indoor air and atmosphere, foodstuff, other materials, etc.	The article focuses on biomonitoring data rather than establishing if and in what quantities phthalates are released from PVC or other plastics. The study does provide specific exposure assessments for medical devices and medications but that is a much narrower subset of products. Medical device exposures arise during use and not from discarded PVC products.

			Consumer products containing phthalates can result in human exposure through direct contact and use, indirectly through leaching into other products, or general environmental contamination. Humans are exposed through ingestion, inhalation, and dermal exposure during their whole lifetime, including intrauterine development.	
§ I.B.2.a. Nature of the Toxicity Presented by Constituent Chemicals				
30.	§ I.B.2.a., p. 9 FN 80 – Mulder (2001)	Researchers, regulators and industry representatives have long known that vinyl chloride causes cancer in laboratory animals and human beings.	At the end of the 1960s, P.L. Viola, an Italian physicist and employee of the PVC-producing company Solvay, began research aimed at inducing acro-osteolysis in rats. However, he found that VC caused cancer in their ears and lungs, the first indication that VC was carcinogenic . . . Based on his work, Maltoni began research on the carcinogenity of VC. This project took twelve years, and early results, published in 1974, confirmed Viola's findings: VC caused angiosarcoma (a rare type of cancer) in the liver, kidneys, and ears of test animals. That same year, B.F. Goodrich made public the fact that three employees had died from angiosarcoma during the past two years. In short order, another eight employee deaths were also ascribed to VC or vinyliden exposure [28–30]. This announcement was a watershed, spurring investigation into the VC problem. The following years were characterized by hectic action from public authorities, industries, labor unions, scientists and the media. In many countries, exposure standards were sharpened (from 500 ppm, to 50 ppm, and ultimately to 1 ppm), and investigations into VC carcinogenity were intensified [28,29]. Worldwide panic ensued. Cancer of the liver had also been detected among employees of PVC processing industries	The history and carcinogenicity of vinyl chloride has been the basis for U.S. and other governmental agencies for more than 40 years. Some language in the article is incorrect, such as "[w]orldwide panic ensued."

			where VC exposure levels were much lower than in PVC production. Furthermore, ordinary citizens also appeared to be at risk: VC had been detected in packaging, food, and drinks, as well as in household goods and furnishing [27,31]. Researchers in the UK concluded that the wives of PVC workers were twice as likely to suffer miscarriages [32]. By June 1974, 21 cases of angiosarcoma had been diagnosed [28] and by 1995, 175 cases of this rare type of liver cancer had been confirmed worldwide [33]. From that point on, manufacturers began to bring down VC concentrations in the workplace and in products, as well as VC emissions from factories (Fig. 4). Generally, these have been under control by the industry since the end of the 1970s, at least in the U.S., Japan, and Western Europe. Automatic reactor cleaning, stopping leaks, closed vessels and personnel training to recognize and treat leaks were among the most important measures.	
31.	§ I.B.2.a., p. 9 FN 81 – Ye (2014)	Inadequate waste management has already contributed to the extensive chemical pollution of the marine environment.	The aim of the present study was to examine whether long-term exposure to DEHP and its active metabolite mono- (2-ethylhexyl)-phthalate (MEHP) disrupts endocrine function in marine medaka (Oryzias melastigma). Di-(2-ethylhexyl)-phthalate (DEHP) has been widely used as a plastic softener in manufacturing of various products composed of polyvinyl chloride (Koo and Lee, 2004) . . . Because DEHP is ubiquitous in the environment, it is expected that its metabolites are also ubiquitous. Mono-(2-ethylhexyl)-phthalate (MEHP) was detected at concentrations of 0.010–1.30 μg/L in the Tama River in Tokyo (Suzuki et al., 2001). In Canada, the MEHP concentrations were	None of the cited locations are in the U.S.

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			45.49–57.2 ng/L, 3.30–6.72 ng/g wet wt, 0.39–1.13 ng/g wet wt, 0.24–1.1 ng/g wet wt and 0.33–0.84 ng/g dry wt in sea water, blue mussels, Dungeness crab, white spotted greenling and sediments, respectively (Blair et al., 2009). The wide distribution of DEHP and MEHP in marine environments has aroused great concern for aquatic organisms.	
32.	§ I.B.2.a., p. 9 FN 81 – Kang (2010)		The present study was conducted to assess the biochemical changes in evaluating toxic effects of DEP in olive flounder (Paralichthys olivaceus), one of the most important aquaculture fishes in far-eastern Asian countries, by measuring several parameters in the liver, kidney and serum after laboratory exposure. In view of the oxidative stress activity of DEP and other phthalates in rats (Seo et al., 2004), anti-oxidant parameters were the focus of the current assessment. DEP is known to be released into the environment during its synthesis processes, final utilization of DEP-containing products, or disposal of used products (Giam et al., 1978; Joblings et al., 1995). The most important release source to the aquatic environment is ascribed to leaching from landfill sites (Silva et al., 2004). Consequently, DEP has been widely identified in aquatic environments and their biota (Giam et al., 1978; Fatoki and Vernon, 1990).	The study includes products and use among plasticizers release sources, and none in the U.S.
33.	§ I.B.2.a., p. 9 FN 82 – Sanders (1973)	Aquatic organisms accumulate phthalate plasticizers directly from the surrounding water, as well as through the consumption of contaminated food and particles, giving rise to significant concerns about	*Parenthetical:* (reporting that "[i]nvertebrates exposed continuously to [phthalate esters in water rapidly accumulated total body residues many times greater than the concentrations in water") Aquatic invertebrates were exposed to di-n-butyl and di-2-ethylhexyl phthalate esters in water to determine toxicity, accumulation,	The study downplays the results of phthalates on aquatic invertebrates themselves, stating that the results "suggest that the compounds might be relatively safe as far as aquatic organisms are concerned." Although the study suggests that phthalate exposure could impact predatory vertebrates higher on the food chain, it does not offer evidence of this hypothesis.

		far-reaching biological consequences.	and reproductive effects of these compounds. Invertebrates exposed continuously to phthalate esters in water rapidly accumulated total body residues many times greater than the concentrations in water . . . However, phthalate residues were not magnified in invertebrates to the same degree as found with organochlorine insecticides. The process of biological magnification of chemical residues in an organism is largely dependent on the balance existing between the process of accumulation and elimination . . . The low degree of toxicity and the high excretion rate of di-n-butyl and di-2-ethylhexyl phthalates suggest that these compounds might be relatively safe as far as aquatic organisms are concerned, provided exposure is not constant. However, successful growth and reproduction are essential for the maintenance of animal populations. Aquatic invertebrates are the main food source of many fishes and wildlife, and thus growth and reproduction of these predatory vertebrates could be adversely affected in an indirect manner whether or not phthalate esters directly affected fish and wildlife. The present evidence indicates that phthalate esters in small amounts are detrimental to ecologically important aquatic invertebrates, and these compounds should therefore be considered as environmental pollutants.	
34.	§ I.B.2.a., p. 9-10 FN 83 – Fossi (2012)	Researchers have hypothesized that endangered whales may be chronically exposed to phthalates	The study examined the impact of microplastics on the Mediterranean fin whale: In view of the presence of microplastics in the Mediterranean environment, the	This study examined water samples from the Mediterranean and fin whales stranded on the coasts of Italy. The study identified the presence of MEHP (a DEHP metabolite) in four out of five stranded fin whales, but there is no direct evidence that this metabolite is present due to the

			detection of plastic additives in the blubber of fin whales and the long lifespan of the species, fin whales appear to be chronically exposed to persistent and emerging contaminants as a result of microplastic ingestion. . . The presence of harmful chemical in Mediterranean fin whales, associated with the potential intake of plastic derivatives by water filter and plankton ingestion, was demonstrated for the first time by the results of this study, which documented the presence of relevant concentrations of MEHP in the blubber of four out of five stranded fin whales (Fig. 1b). MEHP is a marker for exposure to DEHP . . .	uptake of microplastics. The authors hypothesize that this is due to MEHP and DEHP in surface neustonic/planktonic and water column samples – e.g., "The presence of harmful chemicals in Mediterranean fin whales, associated with the *potential* intake of plastic derivatives by water filtering and plankton ingestion . . ."
35.	§ I.B.2.a., p. 10 FN 84 – Oehlmann (2009)	In addition, laboratory evidence links environmentally relevant concentrations of these compounds to behavioral and developmental abnormalities in a range of aquatic species,	*Parenthetical:* ("Exposures to phthalates have . . . been shown to alter behavior in fish.") Exposures to phthalates have also been shown to alter behaviour in fish. Exposure to 100 mg BBP l21, via the water, caused alterations in shoaling and feeding behaviour in three-spined stickleback (Gasterosteus aculeatus) (Wibe et al. 2002, 2004), and exposure to 5 mg DEP l21 caused alterations in the general behaviour of common carp (Barse et al. 2007). It should be noted that these studies have employed very high exposure concentrations, and these are unlikely to occur in the water column in part owing to their low solubility. Exceptions to this may be for fish living in/closely associated with the sediments in heavily contaminated environments. Exposures of fish to lower levels of phthalates have generally found no adverse effects. In the present experiment, "[e]xposure to BBP did not affect the concentration of VTG in the plasma at any of the	The study itself identifies that most cases of altered fish behavior have only been observed when very high exposure concentrations have been employed – concentrations that are unlikely to occur in the water column but may be possible in heavily contaminated sediments. The study results are for BBP only.

			concentrations tested in males . . . Similarly, there was no evidence of any chemical effect on the number of eggs spawned . . . Furthermore, there were no effects of BBP on the mean percentage viability of embryos at 8 hpf (figure 1). These results are in agreement with reports in the literature, where VTG induction in males, or reproductive effects on females, has been induced only at exposure concentrations that far exceed the measured concentrations in our study. In contrast, however, we found consistent changes in parameters of sperm quality in BBP-exposed male zebrafish. . . . At the highest concentration tested (15 mg BBP l21), the average curvilinear velocity of the sperm was restored to levels similar to those found in the water control tanks (31 mm s21; figure 2a)."	
36.	§ I.B.2.a., p. 10 FN 84 – Ye (2014)		*Parenthetical:* (explaining that "DEHP has been extensively characterized as a developmental and reproductive toxicant in many aquatic toxicological studies," and reviewing relevant research) The aim of the present study was to examine whether long-term exposure to DEHP and its active metabolite mono- (2-ethylhexyl)-phthalate (MEHP) disrupts endocrine function in marine medaka (Oryzias melastigma). DEHP has been extensively characterized as a developmental and reproductive toxicant in many aquatic toxicological studies.	
37.	§ I.B.2.a., p. 10 FN 85 – Carnevali (2010)	demonstrating a "concrete risk" for populations living in polluted regions and threatening a cascade of effects throughout the ocean ecosystem.	In this study, we provide evidence that environmentally relevant concentrations [21] of DEHP interfere with zebrafish reproductive performance, representing a concrete risk for the aquatic population living in polluted areas.	In this study, zebrafish were exposed to DEHP at doses of 0.02, 0.2, 2, 20 or 40 μg/l. Although the study refers to these as "environmentally relevant" concentrations no rationale is offered for why that is the case or whether these dosages are representative of actual environmental exposures.

			We found that exposure of female zebrafish to DEHP or EE2 [17a-Ethynylestradiol served as the positive control] led to a significant increase of circulating levels of vitellogenin. Since estrogen is well known for its effect on promoting vitellogenesis, this observation supports our hypothesis that DEHP has estrogenic effects in zebrafish. However, in fish exposed to the highest dose DEHP, there was a significant decrease in the number of post-vitellogenic oocytes and the number of ovulated eggs was dramatically decreased by all treatments. We can conclude that all environmental relevant doses of DEHP affect vitellogenesis, demonstrating its estrogenic potency. Different dose-related effects have been observed in relation to maturation and ovulation process. The lowest doses have a stronger negative effects on signals inducing maturation (LHR and mPRb), while the highest doses have a greater impact on the inhibition of ovulation (ptgs2). The results of this study, both in vivo and in vitro, clearly demonstrate that all doses of DEHP strongly impair oocyte maturation and ovulation by influencing the expression of factors involved in these processes.	
38.	§ I.B.2.a., p. 10 FN 85 – Sanders (1973)		Aquatic invertebrates were exposed to di-n-butyl and di-2-ethylhexyl phthalate esters in water to determine toxicity, accumulation, and reproductive effects of these compounds. The low degree of toxicity and the high excretion rate of di-n-butyl and di-2-ethylhexyl phthalates suggest that these compounds might be relatively safe as far as aquatic organisms are concerned, provided exposure is not constant. However, successful growth and	As indicated above, this study seems to downplay the results of phthalates on aquatic invertebrates themselves, stating that the results "suggest that the compounds might be relatively safe as far as aquatic organisms are concerned."

			reproduction are essential for the maintenance of animal populations. Aquatic invertebrates are the main food source of many fishes and wildlife, and thus growth and reproduction of these predatory vertebrates could be adversely affected in an indirect manner whether or not phthalate esters directly affected fish and wildlife. The present evidence indicates that phthalate esters in small amounts are detrimental to ecologically important aquatic invertebrates, and these compounds should therefore be considered as environmental pollutants.	
39.	§ I.B.2.a., p. 10 FN 86 – Ye (2014)	Relative salinity may influence the toxicity of aquatic contaminants.	Aquatic toxicological investigations of DEHP were mostly limited to freshwater species; to our knowledge, the effect of DEHP on marine fish has not been reported. However, salinity has been shown to influence the toxic effects of chemicals in aquatic organisms by changing the characteristics of the chemicals or the physiological characteristics of the aquatic organisms. Thus, the effect of a given chemical on marine fish is likely to be different from its effect on freshwater fish. Therefore, a marine fish model for assessing the toxicity of DEHP in the marine environment is urgently needed. Taken together, the results obtained in this study demonstrate that exposure of marine medaka to DEHP and MEHP from hatching to adulthood causes endocrine disruption, disturbs the sex hormone balance, induces liver VTG in males, and alters gonad histology and the transcriptional profiles of key genes along the HPG axis in a sex-specific manner. These alterations subsequently cause reproductive impairment, promote the sexual maturation and impair the fecundity of female medaka, and decrease the fertilization capacity of male medaka. This is the first report	This article states that it is "the first report indicating that MEHP induces endocrine disrupting effects in marine aquatic organisms."

			indicating that MEHP induces endocrine disrupting effects in marine aquatic organisms; the toxic effects of DEHP were induced by DEHP itself and DEHP metabolites, including MEHP.	
40.	§ I.B.2.a., p. 10 FN 87 – Ghorpade (2002)	Thus, although phthalate exposure alters enzyme activity in the vital organs of certain freshwater fish, contributing to "sluggish, non-motile behavior," these compounds appear to produce opposite effects among some brackish species.	DEP is known to be a contaminant of freshwater and marine ecosystems. Therefore, a study was designed to determine the toxic e4ects of DEP on a freshwater fish, *Cirrhina mrigala*. The fish was treated with 25, 50, 75, and 100ppm (w/v) DEP dissolved in acetone to determine the LC50. Positive controls were treated with acetone only. There was 100% mortality observed within 24h in 75 and 100ppm, and 50% mortality in 50ppm treated fish in 72h. Those treated at 25ppm showed only 10% mortality within 72h and remaining fish continued to survive. The surviving fish were treated with 25ppm DEP once daily for 3 days with every change of water (Group III). One group was maintained as negative control in dechlorinated water (Group I) and the other group received acetone once daily for 3 days with every change of water and was used as positive control (Group II). It was observed that soon after the addition of DEP into the tanks the fish became sluggish for approximately 8h. They would confine themselves to one corner of the tank and quietly remain there at the bottom. This behavior was not observed in the positive and negative controls. The AchE in the brain of DEP-treated fish was found to be significantly decreased, indicating that DEP inhibits AchE activity. This correlates well with the sluggish, non-motile behavior of the DEP-treated fish. AchE activity in the muscle of DEP-treated fish did not change significantly. This could be due to the lipophilic nature of DEP,	Given the exposure levels, this laboratory study of DEP is not a basis for assuming effects from discarded PVC products.

			which may be taken up much faster by brain tissue than by muscle tissue. Hence, immediately after addition of DEP to the tanks, the fish were inactive for quite some time.	
41.	§ I.B.2.a., p. 10 FN 88 – Kaplan (2013)	Specifically, a recent study found that mummichogs (Fundulus heteroclitus) were approximately twice as likely to engage in agitated swimming patterns after brief exposure to low phthalate concentrations.	Since low-level environmental contamination may disrupt shoaling and negatively impact both fitness and transmission of social information (Reader et al., 2003), this study examined the influence of BBP on shoaling behavior in F. heteroclitus to determine if the model (fish and behavior) can be utilized as a bioindicator of sub-lethal BBP exposure. In the presence of a predatory stimulus, body size appears to be a primary factor in determining shoal choice, as Fundulus diaphanous prefer to shoal with similarly sized fish irrespective of shoal size or species composition (Krause and Godin, 1994). This study indicates that BBP exposure may significantly disrupt this shoaling behavior in Fundulus. F. heteroclitus focal fish exposed to BBP aggregated with the familiar size-assortative shoal (SLF) significantly less often as compared to either control group. They chose the shoal of small fish approximately five times more often and the neutral zone approximately 2.5 times more often as compared to control. As shoaling appears to provide an anti- predator function for some fishes (Peuhkuri, 1997; Peuhkuri et al., 1997; Pitcher and Parrish, 1993; Ranta et al., 1992), F. heteroclitus selection of either the shoal of small fish or the neutral zone might be viewed as potentially mal-adaptive in terms of protection from predation. . . . This study demonstrates that shoaling and circumspect behaviors among F. heteroclitus individuals were negatively impacted after a relatively short exposure	A laboratory study of BBP.
42.	§ I.B.2.a., p. 10 FN 89 – Kaplan (2013)	In addition, the contaminated individuals exhibited altered social behavior, tending to shoal with relatively small fish, rather than joining similarly sized conspecifics.		
43.	§ I.B.2.a., p. 10 FN 90 – Kaplan (2013)	Because shoaling helps fish to evade predation, while also minimizing competition for food, these effects have "serious negative implications" for exposed individuals and, ultimately, may threaten population viability.		

			period to sub-lethal doses of benzyl butyl phthalate (BBP). [approximate dosage of 0.1 mg/L BBP.] . . . It is unclear at this time if this behavioral disruption in F. heteroclitus is transient or permanent. Further studies are needed to assess potential recovery following cessation of BBP exposure. Nonetheless, individuals with BBP-induced alteration of behavior may no longer benefit from key ecological behaviors that convey an anti-predator effect or minimize competition for food.	
44.	§ I.B.2.a., p. 10 FN 91 – Martinez-Arguelles (2013)	A considerable body of scientific literature indicates that phthalates are potent endocrine disruptors,	In utero exposure to DEHP decreases circulating testosterone levels in the adult rat. In addition, DEHP reduces the expression of the angiotensin II receptors in the adrenal gland, resulting in de- creased circulating aldosterone levels. The latter may have important effects on water and electrolyte balance as well as systemic arterial blood pressure. Therefore, we determined the effects of in utero exposure to DEHP on systemic arterial blood pressure in the young (2 month-old) and older (6.5 month-old) adult rats. Di-(2-ethylhexyl) phthalate is a plasticizer used industrially to add flexibility to polyvinyl chloride (PVC) polymers. Multiple studies have identified DEHP as an endocrine disruptor with antiandrogenic activity (Shelby, 2006).	In utero toxicity of DEHP in rats.
45.	§ I.B.2.a., p. 10 FN 92 – Abdul-Ghani (2012)	which interfere with hormone regulation and reduce reproductive success among multiple aquatic and terrestrial species, including human beings.	*Parenthetical*: (observing that “phthalate-induced DNA damage [among fetal chicks was] consistent with those found in mice and humans”) Fertile eggs were injected with DEHP and morphological, bio-chemical and DNA and behavioral alterations were ascertained at hatching age. To generalize the results,	A DEHP study to “investigate an avian [chick] model for phthalate teratogenicity and neurobehavior teratogenicity.”

			major changes were replicated with another phthalate, DBP. A better comparison can be made with certain cellular parameters where our findings of phthalate-induced DNA damage were consistent with those found in mice (Martino-Andrade and Chahoud, 2010; Takagi et al., 1992) and humans (Duty et al., 2003). Consistently, phthalate-induced behavioral deficits, although of a different nature, have been found both in our chick model (imprinting) and in humans (ADHD) (Engel et al., 2010).	
46.	§ I.B.2.a., p. 10 FN 92 – Swan (2005)		*Parenthetical*: ("[O]ur data suggest that the end points affected by . . . phthalates are quite consistent across species."). In light of the toxicologic literature for MBP, MBzP, and MiBP (Ema et al. 2003; Foster et al. 1980, 1981; Gray et al. 2000; Nakahara et al. 2003), our data suggest that the end points affected by these phthalates are quite consistent across species. A boy with short AGI has, on average, an AGI that is 18% shorter than expected based on his age and weight as well as an increased likelihood of testicular maldescent, small and indistinct scrotum, and smaller penile size. These changes in AGD and testicular descent are consistent with those reported in rodent studies after high-dose phthalate exposure (Ema et al. 2003; Gray et al. 2000; Mylchreest et al. 2000). The lack of association for MCPP [*metabolite of di-n-octyl phthalate*] and MMP [*monomethyl phthalate, metabolite of dimethyl phthalate*], which have not been widely studied, is not inconsistent with the toxicologic literature. This is the first study to look at subtle patterns of genital morphology in humans in relation to any prenatal exposure. . .We	"This is the first study to look at subtle patterns of genital morphology in humans in relation to any prenatal exposure."

			report that AGD, the most sensitive marker of anti- androgen action in toxicologic studies, is shortened and testicular descent impaired in boys whose mothers had elevated prenatal phthalate exposure. These changes in male infants, associated with prenatal exposure to some of the same phthalate metabolites that cause similar alterations in male rodents, suggest that commonly used phthalates may undervirilize humans as well as rodents.	
47.	§ I.B.2.a., p. 10-11 FN 93 – Corradetti (2013)	For example, environmentally relevant concentrations of phthalate plasticizers exert estrogenic effects in zebrafish (Danio rerio), quickly compromising reproductive cell development and thereby impairing fecundity.	In the present study, DEHP – at environmentally relevant concentrations (0.2 and 20mg\L) – was found to interfere with the reproductive performance of adult male zebrafish. In addition to impairing fecundity, DEHP also affected gametogenesis and the integrity of DNA in germ cells. However, following exposure to concentrations of DEHP at 0.2 and 20mg/L, male reproductive capacity recovers in untreated water after 9 and 13 days, respectively (Fig. 6). This suggests that the effects of DEHP are short-lived and might be reversible. In conclusion, these results demonstrate that spermatogenesis in zebrafish may be temporarily impaired by short-term exposure to environmentally relevant concentrations of DEHP.	Zebrafish reproductive performance effects were determined to be temporary and reversed by a break in exposure.
48.	§ I.B.2.a., p. 10-11 FN 93 – Carnevali (2010)		In this study, we provide evidence that environmentally relevant concentrations [i.e., 0.02, 0.2, 2, 20, and 40 μg/l] [21] of DEHP interfere with zebrafish reproductive performance, representing a concrete risk for the aquatic population living in polluted areas.	Carnevali reported that "in fish exposed to the highest dose DEHP, there was a significant decrease in the number of post-vitellogenic oocytes and the number of ovulated eggs was dramatically decreased by all treatments." The highest dose of "environmentally relevant" concentrations is 40 μg/l. This is higher than the "environmentally relevant" dosages tested by Corradetti above.
49.	§ I.B.2.a., p. 11 FN 93 – Sanders (1973)		*Parenthetical*: (concluding that chronic exposure to low phthalates concentrations significantly reduces rates of reproduction among ecologically important aquatic invertebrates).	Similar to Corradetti, who concluded that the effects of DEHP were only temporary, Sanders also concludes that DEHP (and DBP) are relatively safe, provided that exposure is not constant. Although Sanders does not appear to have tested this with an eye to reproduction specifically, based on the

			Continuous exposure of waterfleas for a complete life cycle (21 days) to 3, 10, and 30 µg/liter of di-2-ethylhexyl phthalate in an intermittent-flow system significantly ($P<0.01$) reduced reproduction (Table 3). Total production of off-spring was inhibited 60, 70, and 83 percent in the three concentrations respectively. The degree of reproductive inhibition remained relatively constant during the 21-day exposure period in all concentrations except for the 10 µg/liter treatment where reproduction declined further by 12 percent between 14 and 21 days.	conclusion of the study it seems that a drop in exposure would return the waterfleas to normal reproductive behavior.
50.	§ I.B.2.a., p. 11 FN 94 – Carnevali (2010)	These results are particularly alarming because the genetic structure of zebrafish closely mirrors that of human beings.	Recently, the zebrafish and human genomes have been shown to share extensive conserved syntenic fragments and many zebrafish genes and their human homologs display structural and functional similarities.	
51.	§ I.B.2.a., p. 11 FN 95 – Martinez-Arguelles (2013)	Similarly, low phthalate doses, corresponding to observed levels of human exposure,	In the general population, the majority of DEHP exposure is dietary, particularly fatty foods including dairy, fish, meat, and oils (Shelby, 2006). In these food groups, DEHP levels can reach 20 mg/kg and serve as sources that account for human consumption and exposure (Shelby, 2006). This is in addition, to specific medical exposures where DEHP intake can be as much as 20 mg/kg/day (Loff et al., 2000). The range of human phthalate exposure corresponds to 0.18 to 62.5 mg/kg/day rat exposures when calculating the human equivalent doses (Reagan-Shaw et al., 2008).	Unclear whether non-medical exposure is from discarded PVC products
52.	§ I.B.2.a., p. 11 FN 96 – Martinez-Arguelles (2013) (Fetal Origin)	lead to abnormal sexual differentiation in laboratory animals when administered during the "sensitive window" of fetal development.	The effects of fetal exposure to phthalates on the male reproductive system were unequivocally shown on animal models, principally rodents, in which short term deleterious reproductive effects are well established. By contrast, information on the long term effects of DEHP in utero	Based on the prior Martinez-Arguelles article (FN 95), the testosterone reductions were not triggered until the exposure largely exceeded the upper levels of observed human (and corresponding rat) exposure.

			exposure on gonadal function are scarce, while its potential effects on other organs are just starting to emerge. The present review focuses on these novel findings, which suggest that DEHP exerts more complex and broader disruptive effects on the endocrine system and metabolism than previously thought. In many studies of the endocrine disruptor effects of DEHP, multiple dose regimens have been tested. In general, DEHP exposures occurring before the maturity of the rats, and particularly during the fetal period, increase the chances of long-lasting endocrine disruption [104]. Interestingly, phthalates were identified as antiandrogens [105–107] because of the similar, but not identical, effects of the antiandrogens linuron [108,109], flutamide, and vinclozolin [110]. . . We have used a model in which pregnant Sprague-Dawley rats are gavaged with increasing doses of DEHP from GD14 until birth. This treatment modality resulted in reduced testosterone formation by the fetal testis due to reduced expression of enzymes and proteins involved in steroidogenesis [111] in agreement with other studies [107,112]. Using this model, we also observed that the in utero exposure to DEHP also resulted in decreased testosterone levels in the adult male offspring, with testosterone levels about half of those found in the controls [43,111,113]. The testosterone decrease was triggered when exposure levels were between 50 and 100 mg/kg/day.	
53.	§ I.B.2.a., p. 11 FN 96 – Fisher (2004)		There is the potential for endocrine disrupting chemicals (EDCs) to act at any level of the HPG axis but there is general support for the view that the development and programming of the axis during fetal	Although the study found that exposure to low doses of DEHP (and other chemicals) alone resulted in disruptions in neurodevelopment related to hyperactivity, this is not the case in a mixture group. In a mixture the compounds have a cancelling effect. As such, it does not seem plausible that

			life could be the most sensitive window to permanently alter the homeostatic mechanisms of the endocrine system (IPCS 2002). There is currently no strong data to suggest that environmental EDCs are responsible for the observed degeneration in human male reproductive health. . . The spectrum of disorders induced by phthalate administration to rats closely parallels those observed in human TDS (with the exception of germ cell cancer). . . . These studies support the hypothesis that all of these disorders (low sperm counts, cryptorchidism, hypospadias and testicular cancer) are associated with TDS. The in utero administration of DBP to rodents during the sensitive period of tissue morpho- genesis permanently alters the testis and produces foci of testicular dysgenesis (immature seminiferous tubules with undifferentiated Sertoli cells, SCO tubules, Leydig cell hyperplasia, morphologically distorted tubules and the presence of abnormal germ cells) which persist in the adult animal (Fisher et al. 2003). . . . Definitive data to determine whether human male reproductive health is declining are still required however; the hypothesis of a 'testicular dysgenesis syndrome' is an important advance and may aid our understanding of the underlying aetiology of these disorders. Within the reproductive tract, the male is exquisitely vulnerable to the effects of anti-androgens during development due the reliance on the synthesis and action of androgens for the masculinization of the male reproductive tract. The ability of phthalates to suppress androgen synthesis during development and to induce testicular dysgenesis together with cryptorchidism and hypospadias has close	these effects would be observed in the real world as humans are – as CBD argues itself – exposed to multiple phthalates and other endocrine disruptors at a time.

			parallels with human TDS. However, the crucial question regarding whether the level of environmental chemicals is sufficient to impact on human male reproductive health remains unanswered, although advances will be made from studying the effects of multi-component EDC mixtures in both in vitro and in vivo test systems.	
54.	§ I.B.2.a., p. 11 FN 96 – Tanida (2009)		*Parenthetical*: (reporting that "pre- and neonatal exposure to [endocrine disruptors] can disturb development even though the amounts of exposure are lower than the no-observed-adverse-effect level determined by toxicological tests using adult animals"). Because fetuses and neonates are highly sensitive to physiologically active agents (Warita et al., 2008) and because such chemicals transfer from the mother to her offspring through the placenta and lactation (Mori, 2001; Kobayashi et al., 2002), pre- and neonatal exposure to EDs can disturb development even though the amounts of exposure are lower than the no-observed-adverse-effect level (NOAEL) or the lowest-observed- adverse-effect level (LOAEL) determined by toxicological tests using adult animals.	
55.	§ I.B.2.a., p. 11 FN 97 – Martinez-Arguelles (2013) (Fetal Origin)	Among male offspring, prenatal phthalate contamination causes genital malformations, including undescended testicles and urethra displacement, impaired sperm production and significantly reduced testosterone levels, which persist into adulthood.	We have used a model in which pregnant Sprague-Dawley rats are gavaged with increasing doses of DEHP from GD14 until birth. This treatment modality resulted in reduced testosterone formation by the fetal testis due to reduced expression of enzymes and proteins involved in steroidogenesis [111] in agreement with other studies [107,112]. Using this model, we also observed that the in utero exposure to DEHP also resulted in decreased testosterone levels in the adult male offspring, with testosterone levels about half of those found in the controls [43,111,113]. The testosterone decrease was triggered when exposure levels were	The testosterone reductions observed here do not appear to be triggered until the exposure largely exceeded the upper levels of observed human (and corresponding rat) exposure based on the prior Martinez-Arguelles article (FN 95).

			between 50 and 100 mg/kg/day. Interestingly, the decrease was in the presence of near normal Leydig cell numbers, as well as near normal proteins and enzymes involved in the transport of cholesterol into the mitochondria and in downstream steroidogenic enzymes, which suggested that the classical steroidogenic pathway was not affected in the adult offspring [111,113]. These long-term anti-androgenic effects also were observed with the phthalate DBP, suggesting a similar mechanism of action between phthalates [114]. In the	
56.	§ I.B.2.a., p. 11 FN 97 – Frederiksen (2007)		Several animal studies have demonstrated that perinatal exposure to DBP and the more long-branched phthalates such as BBzP, DEHP and DiNP and their metabolites results in altered sexual differentiation in male rats. The effects observed includes cryptorchidism, decreased testosterone levels, testicular atrophy, Sertoli cell abnormalities, decreased weight of the androgen-dependent organs, reduction in daily sperm production and lower epididymal sperm counts [2, 32, 74 – 79]. Exposure to the short-branched phthalates, DMP and DEP, has not shown similar effects [75]. Whether phthalate exposure also represents a risk for human health is still unclear as human studies have not yet shown a clear causative link between phthalate exposure and human health problem [81]. However, recent research in this area has indicated that phthalate exposure may also be harmful to humans. Two human studies have related health outcomes to foetal and neonatal exposures to phthalates [20, 26, 88] . . . All in all, these correlations pointed to a decreased androgen action in the boys with the highest	As noted in the study, several animal studies have demonstrated that perinatal exposure to (as relevant here) DBP, DEHP and DiNP have impacted the sexual development of male rats; however, human studies have yet to show clear *causative* link between phthalate exposure and human health problems.

			maternal expo- sures. Data from the two human studies are summarised in Table 5.	
57.	§ I.B.2.a., p. 11 FN 97 – Latini (2006)		Here we review the data that support or discounts the evidence existing to date linking phthalate exposure and the decline of human male fertility, especially in developed countries. Many adverse effects on animal fertility and repro- duction have been documented for phthalates following exposure before puberty. In particular, certain phthalate esters (DEHP, DBP, BBP) when administered to pregnant experimental animals during a critical window of development appear to play a relevant role in determining reproductive and developmental toxicity (Ablake et al., 2004; Calafat et al., 2004; Carruthers and Foster, 2005; Ema and Miyawaki, 2002; Foster et al., 2000; Kai et al., 2005; Latini et al., 2004a,b; Lehmann et al., 2004; Moore et al., 2001; Mylchreest et al., 1998, 2000; Parks et al., 2000; Thompson et al., 2004; Wilson et al., 2004; Zhang et al., 2004). These esters have been shown to produce a syndrome of reproductive abnormalities. The "phthalate syndrome" is characterized by malformations of the epididymis, vas deferens, seminal vesicles, prostate, external genitalia (hypospadias), cryptorchidism and testicular injury together with permanent changes (feminization) in the retention of nipples/areolae (sexually dimorphic structures in rodents) and demasculinization of the growth of the perineum resulting in a reduced anogenital distance (AGD), i.e. the distance from the anus to the base of the scrotum in males and from the anus to the base of the genitals in females . . . Critical to the induction of these effects is a considerable reduction in fetal testosterone synthesis and subsequently androgen levels	This study describes parallels between the effects seen in testicular dysgenesis syndrome (TDS) in humans and "phthalate syndrome" in animals following exposure to phthalates, but states that there is no causal relationship between exposure to phthalates in humans and these effects.

			at the critical window for the development of the reproductive tract normally under androgen control, thus disrupting male reproductive tract organo- genesis (Foster, 2005, 2006; Mylchreest et al., 1999; Parks et al., 2000). It is well known that impairment of fetal testicular testosterone production, or blockade of the androgen receptor leads to an incomplete virilization of the male reproductive tract and reduced fertility (Bowman et al., 2003; Gray et al., 1999; Liu et al., 2005; Mylchreest et al., 1999; Parks et al., 2000). Although no cause and effect relationship exists after exposure of humans to phthalates, the "phthalate syndrome" has parallels with the testicular dysgenesis syndrome (TDS) in humans, which includes interrelated disorders, such as low sperm counts, hypospadias, cryptorchidism and testicular germ cell cancer and may develop during fetal life under the influence of environmental factors	
58.	§ I.B.2.a., p. 11 FN 98 – Martinez-Arguelles (2013) (Fetal Origin)	Exposed females may experience excessive breast tissue growth and altered fertility cycles, in addition to various reproductive organ abnormalities	In humans, studies have shown an association between high levels of MEHP and an increase in early pregnancy loss [152], decreased gestation times [16,153], and endometriosis [154]. Premature breast development has also been reported in a Puerto Rican cohort [155] but this finding has been disputed because of the unusually high levels of phthalates reported and the lack of in vivo evidence sup- porting the proestrogenic and developmental effects required for premature thelarche [156]. A common finding among studies is the fact that single urine measurements were used to correlate the findings with MEHP exposure. However, this is only representative of acute exposure; perhaps, stronger associations would be found by characterizing bioexposure with multiple urine measurements.	The findings regarding breast tissue growth are tied to a very specific cohort (Puerto Rican) with unusually high phthalate exposure. The article indicates that many of the studies on females use only a single urine measurement to correlate findings with MEHP (DEHP metabolite) exposure, deemed insufficient to form the basis for strong associations between exposure and observed impacts.

59.	§ I.B.2.a., p. 11 FN 99 – Ye (2014)	Recent laboratory studies demonstrate that phthalate metabolites induce similar or more severe effects among marine animals	Taken together, the results obtained in this study demonstrate that exposure of marine medaka to DEHP and MEHP from hatching to adulthood causes endocrine disruption, disturbs the sex hormone balance, induces liver VTG in males, and alters gonad histology and the transcriptional profiles of key genes along the HPG axis in a sex-specific manner. These alterations subsequently cause reproductive impairment, promote the sexual maturation and impair the fecundity of female medaka, and decrease the fertilization capacity of male medaka. This is the first report indicating that MEHP induces endocrine disrupting effects in marine aquatic organisms; the toxic effects of DEHP were induced by DEHP itself and DEHP metabolites, including MEHP.	
60.	§ I.B.2.a., p. 11 FN 100 – Martinez-Arguelles (2013)	and rodents.	Once absorbed, DEHP is metabolized into mono-2-ethylhexyl phthalate (MEHP) by lipases in the lining of the gut. MEHP has antiandrogenic activity 10-times greater than DEHP (Frederiksen et al., 2007).	Although the study makes a statement regarding the impact of MEHP, it is made in the context of a discussion regarding human impacts – not rodents. The study examines the effects of DEHP on arterial blood pressure, which seems to be an entirely different aspect of potential phthalate impacts than those observed on the reproductive system.
61.	§ I.B.2.a., p. 11 FN 101 – Swan (2010) (Masculine Play)	In the United States and other industrialized nations, human exposure to multiple phthalate plasticizers is "virtually universal,"	Although several of these are now banned for use in toys and some other products designed for young children (Kamrin, 2009), this legislation does not limit prenatal exposure. Moreover, phthalates are present in so many other products and manufactured in such quantity, that exposure is virtually universal (CDC, 2005).	The article does not establish a connection between discarded PVC products and phthalate exposure.
62.	§ I.B.2.a., p. 11 FN 101 – Howdeshell (2008)		The current review will focus on the effects of in utero exposure to phthalate esters on male rat reproductive tract development and function when administered during the period of sexual differentiation. We will review what is understood about the cellular and molecular mode of action behind phthalate action during reproductive tract development from studies on the well-	Is not focused on exposure from PVC products.

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			characterized phthalate esters di(n)butyl phthalate (DBP, CAS RN 84-74-2) and diethylhexyl phthalate (DEHP, CAS RN 117-81-7), including evidence of strain differences in target organ responsiveness to DEHP. Phthalate esters are found in many commonly used products, including children's toys, health and beauty supplies (e.g. cosmetics and perfumes), medical equipment (e.g. dialysis tubing and intravenous bags), and the enteric coating of some pharmaceuticals. As humans are exposed to multiple phthalate esters on a daily basis, we summarize our research on the cumulative effects of binary and complex mixtures of phthalates with each other and/or with other androgen-disruptive chemicals on male reproductive development. . .	
63.	§ I.B.2.a., p. 11 FN 102 – Frederiksen (2007)	beginning in the womb	Considering that phthalates may be important endocrine disrupters for humans, particularly in foetal life and early childhood, it seems most important to identify the metabolic pathways of phthalates from the first host, the mother, to the second host, the foetus and infant through placenta and breast milk (Fig. 3). Little is known about these path- ways, but recent studies have confirmed that unborn and infants are in fact highly exposed to phthalates. An American study of samples from 54 anonymous donors has shown very low excretion of phthalate metabolites into amniotic fluids. In fact, out of ten measured phthalate metabolites only MEP, MPB and MEHP were found in amniotic fluid and the maximum levels of these three compounds were 9.0, 264 and 2.8 ng/mL, respectively [47].	This study confirms that fetuses are known to be highly exposed to phthalates; however, it also discusses an American study that only detected low levels of phthalate metabolites in amniotic fluid. It is unclear whether this is indicative of exposure or is geared more towards discerning the fetal exposure pathway.

64.	§ I.B.2.a., p. 11 FN 102 – Martinez-Arguelles (2013)		*Parenthetical*: (explaining that phthalates and their metabolites are present in amniotic fluid, umbilical cord blood and breast milk). We determined the effects of in utero exposure to DEHP on systemic arterial blood pressure in the young (2 month-old) and older (6.5 month-old) adult rats. Sprague-Dawley pregnant dams were exposed from gestational day 14 until birth to 300 mg DEHP/kg/day. DEHP and its metabolites have been found in semen (Phillips and Tanphaichitr, 2008), saliva (Silva et al., 2005), amniotic fluid (Huang et al., 2007; Silva et al., 2004), umbilical cord blood (Latini et al., 2003), human milk and baby formula (Frederiksen et al., 2007; Huang et al., 2009).	The language that CBD relies on is only offered in the introduction of this study to set the stage for why the researchers are investigating the effects of in utero DEHP exposure on blood pressure. The study itself does not provide data or evidence of the commonality of DEHP exposure in the womb. To that end, the cited language is only relevant to DEHP and not the remaining phthalate plasticizers identified in the petition.
65.	§ I.B.2.a., p. 11-12 FN 103 – CDC (2013)	And continuing throughout life	CDC researchers found measurable levels of many phthalate metabolites in the general population. This finding indicates that phthalate exposure is widespread in the U.S. population.	This biomonitoring study examines human exposure to phthalate metabolites but states that "[m]ore research is needed to assess the human health effects of exposure to phthalates."
66.	§ I.B.2.a., p. 12 FN 104 – Stahlhut (2007)	thereby raising concerns about negative health consequences at every age.	*Parenthetical*: (explaining that phthalate exposure might affect different segments of the population differently). To our knowledge, ours is the first human study to examine associations between phthalate metabolites and either abdominal obesity or insulin resistance. . . Like our study, others have found associations with outcomes that might follow from antiandrogenic effects of MBP (Duty et al. 2003; Hauser et al. 2006; Main et al. 2006; Swan et al. 2005) and MEP (Jönsson et al. 2005; Main et al. 2006; Swan et al. 2005), and found no association with MEHP (Duty et al. 2003; Hauser et al. 2006; Jönsson et al. 2005; Main et al. 2006; Swan et al. 2005). Results for MBzP,	The article addresses how phthalate exposure may impact people at various ages.

			MEHHP, and MEOHP were less consistent. Assuming these associations represent true effects, differences between our results and others could be due to our larger sample sizes and increased power. Phthalates might also affect adult males differently than fetuses, or they may interact directly with energy balance or glucose metabolism pathways in addition to anti-androgenic effects.	
67.	§ I.B.2.a., p. 12 FN 105 – Hokanson (2006)	For example, experimental and epidemiological evidence demonstrates that low levels of prenatal phthalate exposure influence fetal hormone regulation, resulting in abnormal development of the brain	Data presented here suggest that in vitro exposure of human cells to the plasticiser DEHP at relatively low concentrations may cause changes in gene expression, that one of the two genes chosen to be corroborated using qrtPCR has been specifically shown to be correlated at haploid levels with failure of the fetal brain to develop properly, and the other is correlated with faciodigitogenital dysplasia. At this time, there are no data to directly correlate down-regulation of FGD1 and LIS1/PA- FAH1B1 in vitro with in utero effects of exposure.	The study concludes that there is no data to directly correlate the down-regulation of FGD1 and LIS1/PA-FAH1B1 – both of which are essential for fetal brain development – in *in vitro* effects of exposure with *in utero* exposures.
68.	§ I.B.2.a., p. 12 FN 106 – Suzuki (2012)	and reproductive organs.	We found that maternal urinary MEHP was significantly inversely correlated with the AGI of male newborns. This result was consistent with previous findings and this consistency suggests that prenatal exposure to DEHP, and its metabolite MEHP, affects reproductive development in human males at current exposure levels.	The conclusions of both studies indicate that the impacts on reproductive organs were limited to males that were exposed to phthalate metabolites in utero – as opposed to humans generally. Notably, the Swan study included the following disclaimer: "Our analysis is based on a single measure of AGD, and the reliability of this measurement in humans has not been established."
69.	§ I.B.2.a., p. 12 FN 106 – Swan (2005)		We report that AGD, the most sensitive marker of anti-androgen action in toxicologic studies, is shortened and testicular descent impaired in boys whose mothers had elevated prenatal phthalate exposure. These changes in male infants, associated with prenatal exposure to some of the same phthalate metabolites that cause similar alterations in male rodents, suggest that commonly used phthalates may undervirilize humans as well as rodents.	

70.	§ I.B.2.a., p. 12 FN 107 – Swan (2010) (Masculine Play)	Additional adverse effects, such as altered gender-specific play behaviors	As we hypothesized based on anti-androgenic activity reported in many rodent (as well as human) studies, metabolites of DEHP and DBP were most strongly associated with play behaviour in males, whereas other phthalate metabolites (MEP, MCPP, MMP and MBzP) were not, when examined individually.	The study acknowledges that it used a small sample size and that only a single prenatal urine sample was taken from each participating woman late in pregnancy.
71.	§ I.B.2.a., p. 12 FN 108 – Jones (2008)	and the potential for increased susceptibility to drug addiction, may become apparent only later in life.	*Parenthetical:* (explaining that "exposure to environmental estrogens during development can impact adult behaviors and sensitivity to the rewarding effects of drug abuse"). In contrast to its effects on MA and morphine-induced behaviors, BPA treatment resulted in reductions of amphetamine-induced behaviors [90]. Reasons for this discrepancy are unknown and more research is needed to further elucidate any role that BPA may have in the development of drug addiction. Nevertheless, taken together, these results indicate that exposure to environmental estrogens during development can impact adult behaviors and sensitivity to the rewarding effects of drugs of abuse.	The quotation that CBD cites in the parenthetical is associated with the effects of BPA on drug-induced behaviors. This is irrelevant to the petition.
72.	§ I.B.2.a., p. 12 FN 109 – Main (2006)	Ingestion of contaminated breast milk interferes with androgenic hormone production in male infants, potentially affecting sexual development,	We found subtle but significant dose-dependent associations between neonatal exposure to phthalate monoesters in breast milk and levels of reproductive hormones in boys at 3 months of age. The observed effects on endocrine hormone levels could therefore be late effects of fetal exposure or additive fetal and neonatal expo- sure through the mother, or exposure to a home environment generally rich in phthalates during pregnancy and infancy.	As an initial matter, the study used samples of breast milk from a cohort study performed at hospitals in Finland and Denmark and may not be representative of U.S. exposure. The study noted that it could not account for multiple variables in the study – e.g., differentiating between in fetal exposure versus breastmilk exposure, accounting for potential contamination by breast pumps, etc.
73.	§ I.B.2.a., p. 12 FN 110 – Kim (2009)	and childhood exposure may contribute to rising	In this study, we demonstrated that the concentration of urine phthalates was significantly correlated with teacher-rated ADHD symptom scores and with omission	As indicated in the study's conclusion "the results of this study *suggest the possibility* of an association between phthalate metabolites . . . and the inattention and hyperactive-impulsivity phenotype of ADHD. Indeed, the study

		rates of attention deficit hyperactivity disorder,	and commission errors on neuropsychological tests after adjusting for variables including age, gender, IQ, parental educational level, and SES. To our knowledge, this study was the first to show an association between ADHD symptoms (inattention and hyperactivity/impulsivity) and urine phthalates in human subjects. Previous animal studies (6,15,16) have shown that phthalate-related metabolites induce hyperactivity in rats.	acknowledged several limitations to its administration/findings – e.g.,: – the study did not rely on the diagnosis of ADHD, only the symptoms of the disorder; – applied only the teacher rating form & not the parental report form; – the data was cross-sectional and correlational, so no inferences about causality are possible; etc.
74.	§ I.B.2.a., p. 12 FN 111 – Kolarik (2008)	asthma	The main finding of this study is that phthalates in indoor dust could be found in all samples from Bulgarian homes. The second main finding is that there is a significant association between the concentration of DEHP in indoor dust and wheezing among preschool children.	Samples of indoor dust were taken for this study from homes in two Bulgarian cities, where the composition of flooring and wall coverings, toys, cosmetics, etc. may differ from American homes. More importantly, the study is not relevant to the disposal of PVC products.
75.	§ I.B.2.a., p. 12 FN 113 – Teitelbaum (2012)	and obesity,	Our main findings were associations of MEP and low MWP with BMI and waist circumference among overweight children. Of note, MEP was the highest concentration individual metabolite in our study. We did not observe associations between the other phthalates and body size among normal weight girls and boys. The current evidence is not sufficient for attributing a causal effect for phthalates on increased body size, but the urinary concentrations of some phthalate metabolites among children and the epidemic of obesity may merit voluntary reductions in exposure where possible.	The study concluded that the findings were not sufficient to attribute a causal effect for phthalates on increased body size.
76.	§ I.B.2.a., p. 12 FN 114 – Colón (2000)	as well as premature breast development in girls.	We detected high levels of phthalate esters in 68% of the samples from thelarche patients. DEP, DBP, and DEHP were detected in levels ranging from tens of parts per billion (nanograms per milliliter) to units of parts per million (milligrams per milliliter) in case samples. MEHP, one of the major metabolites of DEHP, was	The study examines phthalate exposure and does not identify exposures related to discarded PVC products. Further the authors conclude that "this study cannot be interpreted as the cause of premature thelarche in Puerto Rican girls at present. It may well be that the etiology of the various manifestations of premature sexual development (including thelarche) on this island is multifactorial."

			detected in five case samples (Table 1). The presence of this phthalate ester was not caused by sample contamination, because contamination by a metabolite rarely occurs. We detected DEHP in 14% of the control samples. The concentration of this phthalate in control samples was significantly lower than the levels in study samples (Table 2). The findings of this study cannot be interpreted as the cause of premature thelarche in Puerto Rican girls at present. It may well be that the etiology of the various manifestations of premature sexual development (including thelarche) on this island is multifactorial. Other possible environmental cofactors related to exposure should also be considered, especially those unique to the Puerto Rican environment. The following have already been associated with premature sexual development in Puerto Rico: the presence of anabolic steroids in poultry (6) and consumption of soy-based formula with a high phytoestrogen content by Puerto Rican infants (8). The higher exposure to phthalate esters in the Puerto Rican infant population is supported by the high importation of plastic packaged foods and because Puerto Rico is a tropical island with year-round high temperature and humidity, which promotes closed environments with the frequent use of air conditioners in homes and public buildings (36).	
77.	§ I.B.2.a., p. 12 FN 115 – Tanida (2009)	These results are consistent with laboratory data indicating that low doses of DEHP, including levels not previously associated with any adverse effect, "irreversibly" disturb brain	Exposure to several EDs (87nmol/rat) including BPA, p-octylphenol, nonylphenol, dibutylphthalate, dicyclohexylphthalate, and DEHP in the neonatal period disrupts neurodevelopment related to hyperactivity concomitantly with a lower level of TH-ir within midbrain dopaminergic nuclei in the	This study relates to potential endocrine disruptor effects of several compounds. Although the study found that exposure to low doses of DEHP (and other chemicals) alone resulted in disruptions in neurodevelopment related to hyperactivity, this is not the case in a mixture group. In a mixture the compounds have a cancelling effect. As such, these effects may not be observed in the real world as humans are – as

		development among fetal mice, subsequently inducing hyperactivity.	juvenile period (Ishido et al., 2004, 2005). Our results at least reveal that EDs such as phenols, phthalates, and dioxins below NOAEL or LOAEL can cause the loss of midbrain dopaminergic neurons and/or the decrease of TH- biosynthetic activity related to motor activity in case of exposure during the fetal and neonatal periods, and that these changes do not recover until sexual maturation. This implies that EDs contribute to the incidence of neurodevelopmental disorders such as ADHD. In summary, we have demonstrated that in utero and neonatal exposure to EDs such as low doses of BPA, DEHP, or TCDD alone resulted in irreversible changes in immunohistochemical and presumably functional level within midbrain dopaminergic nuclei of mice, whereas such changes did not appear when the animals were exposed to mixtures of the same chemicals, hypothetically because of counteraction caused by thyroid hormones and/or aryl hydrocarbon receptor-related mechanisms. From the comparison data between brain weights of the mixture groups and those of controls (Table 1), this canceling effect is just one apparent response. These results suggest that the practical effects of exposure to multiple EDs that are ubiquitous in the environment are very unique and elusive, since the effects of EDs on neurodevelopment may apparently disappear, leading us to propose a new facet of responses to pollution from combined EDs. The combined effect of all the chemicals present in the human body must be considered in future risk assessments for human health.	CBD argues – exposed to multiple phthalates and other endocrine disruptors at a time.
78.	§ I.B.2.a., p. 12 FN 115 – Culty (2008)		*Parenthetical:* (reporting that "behavioral and other deficits . . . might occur during	This is a toxicology study regarding effects of exposure to a substance and not potential exposure or effects from discarded PVC products.

			early development if the brain is not exposed to adequate androgen levels”). Taken together, these results suggest that a threshold DEHP level might exist beyond which the deleterious effects on Leydig cell function become permanent, but the present results do not rule out the possibility that the effects of in utero DEHP ultimately are reversible, but not as early as PND60. The importance of this observation is that reductions in testosterone from early development through early adulthood could lead to highly significant physiological deficits, including the behavioral and other deficits that might occur during early development if the brain is not exposed to adequate androgen levels [46, 47]. In conclusion, our data suggest that DEHP, when administered during gestation and depending on its dose, can have effects that persist from the neonatal period to adulthood. At this point, it remains unclear whether DEHP might have significant acute or long-term pathophysiological effects with relevance to humans or wildlife at the doses to which humans are exposed. The findings that several phthalates present in the environment and detected in human fluids, such as DEHP and DBP, appear to share target genes and pathways, however, suggest that these compounds could act additively or synergistically on specific organs or cell types to produce deleterious effects.	The study authors did not reach any firm conclusions based on the evidence. For instance – in contrast to CBD’s statement that low doses of DEHP can irreversibly disturb brain development – the study states that it cannot rule out the possibility that the in utero effects of DEHP are reversible.
79.	§ I.B.2.a., p. 12-13 FN 116 – Hauser (2006)	Among adult men, urinary concentrations of phthalate plasticizers correlate with poor semen quality,	The present study confirms in a larger sample of men an association between MBP and below WHO reference value sperm concentration and motility. This result is consistent with toxicologic studies in laboratory rodents showing that MBP is a testicular toxicant.	This is a toxicology study regarding effects of exposure to a substance and not potential exposure or effects from discarded PVC products. This study found an association between MBP (a metabolite of DBP) and altered male reproductive function but did not find and association between semen quality and DEHP metabolites.

			Although laboratory studies in rats consistently find associations between MEHP and altered male reproductive function, no associations between MEHP and semen parameters were observed either in our previous study or in this larger and more powerful analysis. . . The present study did not find strong dose–response associations of semen quality with any of the DEHP metabolite indices.	
80.	§ I.B.2.a., p. 12-13 FN 116 – Duty (2003)		Our study suggests that some phthalate monoesters, at environmental levels, are associated with lower sperm concentration, lower motility and increased percentage of sperm with abnormal morphology in humans. Specifically, we found dose-response relations of MBP with sperm motility and sperm concentration. There was also a dose-response relation between MBzP and sperm concentration. We also found limited evidence for an association of higher MMP with poor sperm morphology. In addition, our data on MEHP are also consistent with animal studies, which have shown that, although MEHP is a testicular toxicant after gestational or lactational exposures, adult exposures are not associated with testicular toxicity.	This is a toxicology study regarding effects of exposure to a substance and not potential exposure or effects from discarded PVC products.
81.	§ I.B.2.a., p. 13 FN 117 – Stahlhut (2007)	abdominal obesity and insulin resistance.	Our objective in this study was to investigate phthalate exposure and its associations with abdominal obesity and insulin resistance. In conclusion, in this large national cross-sectional sample, several phthalate metabolites showed statistically significant positive correlations with abdominal obesity and insulin resistance in adult U.S. males. If confirmed by longitudinal studies, these associations would suggest that phthalates, a widely used family of chemicals, may contribute to the prevalence of obesity,	This is a toxicology study regarding potential effects of exposure to a substance and not potential exposure or effects from discarded PVC products. This study found a correlation, or association, between exposure to several phthalate metabolites and adult male obesity (MBzP, MEHHP, MEOHP, MEP) and insulin resistance (MBP, MBzP, MEP), but these findings have yet to be confirmed.

			insulin resistance, and related clinical disorders.	
82.	§ I.B.2.a., p. 13 FN 118 – Martinez-Arguelles (2013) (Fetal Origin)	Exposed women may be more likely to suffer pregnancy complications	The present review focuses on these novel findings, which suggest that DEHP exerts more complex and broader disruptive effects on the endocrine system and metabolism than previously thought. In humans, studies have shown an association between high levels of MEHP and an increase in early pregnancy loss [152], decreased gestation times [16,153], and endometriosis [154].	This is a toxicology study regarding potential effects of exposure to a substance and not potential exposure or effects from discarded PVC products. As stated in the article, there is only an *association* between high levels of the phthalate metabolite MEHP, and early pregnancy loss, decreased gestation time, and endometriosis. The article does not offer any evidence of causation.
83.	§ I.B.2.a., p. 13 FN 119 – Svensson (2011)	and contract diabetes.	This exploratory cross-sectional study evaluated the possible association between phthalate exposure and self-reported diabetes among adult Mexican women. Exposure to DEHP might play a role in diabetogenesis. This is an incipient research that deserves further attention and longitudinal studies are warranted to confirm the relationship between exposure to some phthalates and diabetes.	This is a toxicology study regarding potential effects of exposure to a substance and not potential exposure or effects from discarded PVC products. Based on this study, there is no definitive evidence that demonstrates phthalate exposure impacts diabetes.
84.	§ I.B.2.a., p. 13 FN 120 – Rusyn (2012)	In addition, scientific studies indicate that phthalate plasticizers may exert carcinogenic effects in the liver and other organs.	Human data on cancer hazard of DEHP are largely inconclusive, because most studies lack appropriate exposure assessment and only indirect evidence for associations between various cancers and exposure to DEHP has been ascertained. The carcinogenicity database in animals conclusively shows that DEHP causes cancer of the liver in male and female mice and rats [13–16]. Importantly, the toxic and carcinogenic effects of DEHP are not limited to liver. The International Agency for Research on Cancer monograph 101 working group concluded [214] that the human relevance of the molecular events leading to cancer elicited by DEHP in several target tissues	This is a review regarding potential effects of exposure to a substance and not potential exposure or effects from discarded PVC products. This study is limited to examining the potential hazards of DEHP and not phthalate plasticizers generally, as the Petition suggests.

			(e.g., liver and testis) in rats and mice cannot be ruled out and DEHP was classified as possibly carcinogenic to humans (Group 2B).	
§ I.B.2.b. Concentration of Toxic Constituents in Discarded PVC				
85.	§ I.B.2.b., p. 13 FN 121 – Brandt-Rauf (2012)	As a whole, the PVC industry consumes over 98 percent of global vinyl chloride production and at least 90 percent of phthalate output worldwide,	PVC is polymerized from vinyl chloride (VC) monomer, which is one of the highest production volume chemicals globally with a current annual worldwide demand of approximately 16 billion pounds which is increasing at an approximate 3% annual rate. Up to 98% of VC is used in the production of PVC.	CBD is attempting to draw a linkage between the amount of chemicals used in the manufacture of PVC and the amount of substances that might be released at its end of life. As discussed elsewhere, this is simply not an appropriate approach because the polymerization of PVC will create an inert material that will not release most of the additives. In addition, CBD is trying to draw an inference from global data, rather than U.S. consumption. The referenced article is out of date and provides no suitable framework for analysis of U.S. PVC consumption. As we will also discuss later, CBD simply cannot draw a linkage with regards to degradation or leaching of phthalates from these production numbers. Plasticized PVC (flexible) represents about 25% of U.S. consumption in 2019. *See* Borrelli, de la Cruz, Paradis, *Residual Vinyl Chloride Levels in US PVC Resins and Products: Historical Perspective and Update*, Journal of Vinyl Additive Technology, 65-66 (April 2005) (the VI estimates that the ratio of rigid and flexible PVC has not changed significantly since publication of Borrelli's article). Using the industry resin data referenced from line 177 below, translates to approximately 0.25 x 10,521 = 2,630 million pounds of resin used in U.S. flexible PVC applications. At a 30% plasticizer composition, U.S. plasticizer use in PVC would amount to (0.30 x 2,630)/0.70 = 1,130 million pounds of plasticizer. The VI estimates that approximately 50% of the plasticizer volume used in the U.S. now constitutes non-phthalate types, which means that approximately 565 million pounds of phthalate plasticizers were used in flexible PVC in 2019. Most of these phthalate plasticizers are the high molecular weight variety, which are known to be less volatile thus, making the plasticizers less available for exposure. Of course, as noted above, no plasticizer in rigid PVC which constitutes the bulk of PVC products sold and distributed in the U.S.

86.	§ I.B.2.b., p. 13 FN 121 – Lithner (2009)		In this study, it was investigated if various plastic products emit hazardous chemical substances to water. Two leaching methods (batch and diffusion tests) were used and the leachates were tested for acute toxicity to Daphnia magna. The PVC polymer is made by polymerising vinyl chloride monomers, a substance which is classified as carcinogenic (ECB, 2008). To prevent degradation by heat during processing stabilisers that contain heavy metals (e.g. Pb, Sn, Cd, or Ba/Zn) are needed (Bacalogulu et al., 2001). Several of the used Pb, organotin, Cd and Ba/Zn stabilisers are very toxic to aquatic organisms, and toxic to reproduction (i.e. Pb, organotin and Cd), and mutagenic and carcinogenic (i.e. Cd; ECB, 2008). To make the PVC soft, a high percentage (10–50wt%) of plasticisers is added (Andrady, 2003). Over 98% of all plasticisers are used in PVC, and 95% of all plasticisers are phthalates (OECD, 2004).	As noted above, the reference is outdated and not reflective of actual practices for most PVC produced in the U.S. In addition, the referenced study is a Swedish study that purchased product presumably in Sweden for evaluation. The suitability of test methods conducts
87.	§ I.B.2.b., p. 13 FN 121 – Sass (2005)		*Parenthetical:* (reporting that "[v]inyl chloride . . . is manufactured exclusively for polymerization into [PVC]").	
88.	§ I.B.2.b., p. 13 FN 122 – Brandt-Rauf (2012)	which respectively exceed 16 and 18 billion pounds each year.	PVC is polymerized from vinyl chloride (VC) monomer, which is one of the highest production volume chemicals globally with a current annual worldwide demand of approximately 16 billion pounds which is increasing at an approximate 3% annual rate. Up to 98% of VC is used in the production of PVC.	
89.	§ I.B.2.b., p. 13 FN 123 – Chatterjee (2010)	These substances are essential components of a wide variety of goods, ranging from artificial leather and traffic cones to plastic bags, children's toys and construction supplies.	According to the American Chemistry Council (ACC, formerly Chemical Manufacturers Association) (CMA 1999), the largest use of BBP is in vinyl tile. BBP is also used as a plasticizer in PVC-based food conveyor belts, carpet tiles, artificial leather, tarps, automotive trim, weather stripping, traffic cones, and to a limited	The article references uses and not exposure from discarded PVC products. Use information is dated and does not reflect switch away from phthalates in many product categories.

			extent in vinyl gloves (IPCS 1999). The compound is also a component of some adhesives, cellulose plastics, and polyurethane (IPCS 1999).	
90.	§ I.B.2.b., p. 13 FN 123 – CDC (2009)		There are numerous products that contain phthalates: adhesives; automotive plastics; detergents; lubricating oils; some medical devices and pharmaceuticals; plastic raincoats; solvents; vinyl tiles and flooring; and personal-care products, such as soap, shampoo, deodorants, lotions, fragrances, hair spray, and nail polish. Phthalates are often used in polyvinyl chloride type plastics, such as plastic bags, garden hoses, inflatable recreational toys, blood product storage bags, intravenous medical tubing, and toys (ATSDR, 2001, 2002).	The article discusses uses and not exposure from discarded PVC products.
91.	§ I.B.2.b., p. 13 FN 123 – Wams (1987)		Some 95% of all DEHP is used as a plasticizer in plastics [5]; polyvinylchloride (PVC) utilises 85% of the total production of plasticizers [6] with DEHP being the most important plasticizer for PVC [1]. It is also used as a plasticizer in cellulose ester plastics and synthetic elastomers [1]. Plasticized PVC is used for car seats, furniture, bloodbags and tubes, vinyl floor and wall covering, cables and in foils for a wide variety of purposes, from packaging to building and construction. Apart from its use in PVC, several other applications of DEHP are worth mentioning [7]. Of these, its application as a plasticizer in industrial paints and adhesives and as a dielectric fluid in condensors seem to be the most important. Its use as a plasticizer in paints is apparently diminishing, since modern paints do not contain free plasticizers. A dielectric fluid on the basis of DEHP is sold by General Electric.	The article discusses uses and not exposure from discarded PVC products.
92.	§ I.B.2.b., p. 13 FN 124 – Rahman (2004)	Depending on desired characteristics, phthalate plasticizers may constitute	*Parenthetical:* (explaining that certain medical plastics, such as dialysis tubing,	As indicated in our 2014 comments, this is an inaccurate account of the content of DEHP in medical bags and tubing.

		up to 80 percent of finished PVC products, thereby posing significant risks to human health and the environment.	contain as much as 80 percent DEHP by weight) The PVC used in IV and blood storage bags typically contain 30–40 wt% DEHP and medical tubing such as dialysis tubing may contain as much as 80 wt% DEHP [24].	
93.	§ I.B.2.b., p. 13 FN 124 – SF Dep't of Env't (2008)		*Parenthetical:* (presenting test results indicating that phthalate plasticizers comprise over 77 percent of certain children's toys)	The Consumer Product Safety Improvement Act of 2008 (CPSIA) imposed a permanent ban of children's toys and childcare articles containing concentrations of more than 0.1% of three specified phthalates: di(2-ethylhexyl) phthalate (DEHP); dibutyl phthalate (DBP); or butyl benzyl phthalate (BBP). CPSIA also imposed an interim ban on children's toys and childcare articles containing more than 0.1% of three other specified phthalates: diisononyl phthalate (DINP); diisodecyl phthalate (DIDP); or di-n-octyl phthalate (DnOP). Separately, CPSIA created third-party testing and certification requirements for children's product makers and sellers. Companies are required to test substances that may contain phthalates to demonstrate that they comply with the restrictions. The Consumer Product Safety Commission (CPSC) is overseeing phthalates in consumer products.
94.	§ I.B.2.b., p. 13-14 FN 125 – Am. Med. Ass'n (2001)	For example, over a decade ago, the American Medical Association warned that PVC treatment devices expose critically ill infants to levels of DEHP likely to impair reproductive development.	Whereas di-ethylhexyl phthalate (DEHP) is a plasticizer used in PVC medical devices such as IV bags, blood bags, and medical tubing; and Whereas numerous studies have found that DEHP leaches from PVC medical devices into blood, blood products, and medical solutions; [1,2,3,4] and Whereas an expert panel convened by the National Toxicology Program Center for the Evaluation of Risks to Human Reproduction expressed "serious concern for the possibility of adverse effects on the developing reproductive tract of male infants exposed to [the] very high levels of DEHP that might be associated with intensive medical procedures such as those used in critically ill infants"; [11] and	The VI supported the discontinuation of DEHP-plasticized neonatal medical devices. Indeed, the FDA, as early as 2002, issued draft guidance on medical devices containing DEHP. *See* U.S. FDA, *Medical Devices Made With Polyvinylchloride (PVC) Using the Plasticizer di-(2-Ethylhexyl)phthalate (DEHP); Draft Guidance for industry and FDA* (Sept. 6, 2002).

			RESOLVED, That CMA strongly urges all hospitals to phase out their use of PVC products containing DEHP in Neonatal Intensive Care Units and encourages the use of commercially available alternatives; and, be it further RESOLVED, That CMA calls upon health professionals, especially those involved in the care of critically ill infants, to encourage the institutions with which they are associated to adopt purchasing policies that will lead to the increasing use of non-DEHP medical devices in Neonatal Intensive Care Units.	
95.	§ I.B.2.b., p. 14 FN 126 – Lithner (2009)	In addition, scientific research demonstrates that a number of PVC consumer products, including bath toys and inflatable swim rings, release phthalate plasticizers and other chemical additives to water in concentrations which may produce acutely toxic effects.	Chemical substances leaching from new plastic consumer products to water caused acute toxic effects (immobility) for D. magna in 9 out of 32 products, with 48-h EC50s of leachates ranging from 5 to 80 g plastic material L^{-1}. A compact disc (recordable) was the most toxic plastic product, but the toxicity was (according to the TIE) caused by the silver layer, not the polycarbonate plastic. Plasticized PVC and polyurethane were the only plastic types of the 15 tested ones that displayed toxicity. None of the 32 tested products were particularly acutely toxic since the EC50 concentrations were quite high (5–80g plastic material L^{-1} or above). Compared to pure chemicals a chemical substance is not classified as acute toxic to Daphnia if 48-h EC50 is >0.1 g L^{-1} (UNECE, 2007).	Although the study did show some acutely toxic effects on D. magna (immobility), the study did not find that any of the tested products to be particularly acutely toxic since the EC50 (half maximal effective concentration) were "quite high" at 5-80g plastic material L^{-1} or above. TO that end, the study authors did not do any chemical analyses to determine what chemicals present in the leachate could be responsible for the toxic effects seen. The report does not indicate where these products were manufactured or purchased.
96.	§ I.B.2.b., p. 14 FN 128 – Bidoki (2010)	For instance, although the construction sector accounts for approximately one-half of PVC demand	Despite claims that PVC production negatively affects the natural environment and human health, it is still widely used. At least 50% of the market is driven by the construction/housing industry.	CBD is attempting to argue that the majority of discarded PVC products are unregulated because most of these products come from the building and construction sector, which accounts for approximately 70% of domestic PVC consumption. *See* Amer. Chemistry Council, 2021 Resin Report; *see also* Krock, R., *Update on Vinyl Industry in U.S. and Canada, Book of Papers of IOM3 PVC 2021 Conference* (May 12, 2021) p. 398. As discussed in our letter, the fact

				that these materials are considered nonhazardous by EPA does not mean they are overlooked. Instead, they are determined to be nonhazardous based on RCRA's longstanding system of analysis.
97.	§ I.B.2.b., p. 14 FN 129 – Magdouli (2013)	and one-third of national DEHP consumption, [. . . . an EPA guidance document identifies most industry waste as nonhazardous, and fails to include any instructions for the proper management of discarded PVC]	In USA, the use of the DEHP as plasticizer recorded in 2005 was reported to be 40% for medical devices, 30% for consumer foods, and 30% for construction applications (Bizzari et al., 2007).	This information is dated. Today, the VI estimates that the volume of DEHP consumed in the U.S. has declined significantly, having been replaced by other phthalates and other non-phthalates. The most significant use of DEHP in PVC is in medical applications, with the remainder being in durable goods such as food-contact tubing (e.g., dairy industry applications) and conveyor belts. Still thhere are a myriad of other non-PVC applications. *See e.g., Final Scope of the Risk Evaluation for Di-ethylhexyl Phthalate (1,2-Benzenedicarboxylic acid, 1,2-bis(2-ethylhexyl) ester)*, EPA (Aug. 2020), p. 26-32 (identifying uses of DEHP).
§ I.B.2.c. Migration Potential				
98.	§ I.B.2.c., p. 14 FN 134 – Walter (2011)	Recent scientific evidence demonstrates that PVC pipe, which constitutes a growing percentage of the nation's water system, leaches increasing concentrations of vinyl chloride and other chemical compounds during use and after disposal.	The purpose of this research was to investigate VC levels encountered in drinking water as a result of the use of PVC/ CPVC pipe. Although all values measured in this study are below the EPA's MCL of 2.0 mg/L, many readings, especially at longer times, were quantifiable and, therefore, above the MCL-Goal of 0 mg/L. For new pipe manufactured in the US, long term studies in the lab suggested equilibrium concentrations of about 300 ng/L. In time frames that simulate overnight stagnancy in a home (5e8 h), VC levels varied from below detection (<6 ng/L) to 20 ng/L, with pipe age and/or manufacturer having some effect. Viewing our results in the context of other studies, consider- able differences in VC accumulation can occur across different manufacturers, especially pipe manufactured outside the U.S. and before 1977. Aging effects of pipe were generally negligible, except for an anomalously high level of VC (>200 ng/L in one week) found in one aged pipe with a rust-colored biofilm.	The laboratory studies of PVC/CPVC pipe demonstrated that the material did not exceed EPA's MCL under numerous tested conditions: – Vinyl chloride levels were below the limit of detection using new Schedule 40 PVC for two weeks. Even after 1+ years of incubation vinyl chloride levels remained 6-10x below EPA's MCL of 2 ug/L. – Vinyl chloride levels remained undetectable (LOD 1.8 ng/L) or below the limit of quantification (LOQ 6 ng/L) in new Schedule 40 and Schedule 80 incubations at time scales which approximate water sitting in pipes overnight in a house (5-10 hours). Even after 100 hours of incubation in both PVC types, vinyl chloride levels remained ~100x lower than EPA's MCL (~20 ng/L after 100 hours vs. 2 ug/L EPA MCL). – Aged PVC with and without biofilm present again exhibited undetectable (<1.8 ng/L) or below LOQ (6 ng/L) during simulated overnight incubation time scales. 100 hour incubations resulted in vinyl chloride levels that were 10-100x lower than EPA's MCL. In addition, of the 15 field samples taken (all from house systems employing PVC or CPVC piping), only 3 exhibited a detectable level of vinyl chloride (LOD 1.8 ng/L).

			While it has been shown that PVC and CPVC pipe reactors leach VC in short (up to 7 days) and long term (up to 2 years) studies, this research provided preliminary evidence that chlorine may also contribute to VC accumulation via DBP reactions. This is supported by the fact that CPVC reactors accumulated more VC in chlorinated reactors compared to dechlorinated reactors over time, indicating that the chlorine in the water, in addition to leaching from the plastic piping, may contribute to VC accumulation.	
99.	§ I.B.2.c., p. 14 FN 134 – Stern (2008)		Few studies available in the open literature comprehensively characterize the leaching of monomers and/or additives from PVC and CPVC polymer materials. "Early-era"2 PVC potable water pipes tended to leach vinyl chloride monomer (VCM) into drinking water. Following the scientific evidence demonstrating that this monomer was carcinogenic in both humans and animal models, new guidelines limiting the amount of vinyl chloride residues permitted in PVC pipes were established by the National Sanitation Foundation (NSF) in 1976. However, pipes manufactured prior to the implementation of the new manufacturing process remain in use in many public water systems and may continue to leach VCM into drinking water. Degradation associated with the age of the tubing and/or predisposing environmental conditions such as high temperatures (Al-Malack and Sheikheldin 2001) may increase the degree to which vinyl chloride residues migrate into water. Al-Malack (2001) studied the effect of water quality parameters on the leaching of lead and other metal heat stabilizers from	The Stern article relies predominantly on foreign studies or studies using foreign-manufactured materials to support its contentions regarding leachate from PVC piping. There is no indication that these pipes meet NSF 61, the standard employed in the U.S. In fact, the presence of lead in PVC piping would automatically disqualify the piping from NSF 61 certification. Further, to the extent that 'early-era' piping is still in use, vinyl chloride leaching would be limited to small diameter pipe (i.e., less than 2 inches). Since this small diameter piping was not commonly used at the time, its prevalence today would be very limited. Carroll, Jr, William & Eckstein, Dave, *Vinyl chloride in rural water from pre-1977 applications of PVC pipe.* Journal of Vinyl and Additive Technology. Vol. 7 (2001) p. 49, Table 1; *see also* Sustainable Solutions Coalition *Life Cycle Assessment of PVC Water and Sewer Pipe and Comparative Sustainability Analysis of Pipe Materials (2018), https://www.uni-bell.org/files/Reports/Life_ Cycle_Assessment_of_PVC_Water_and_Sewer _Pipe_* and_Comparative_Sustainability_Analysis_of_Pipe _Materials.pdf (stating that, "EPA has found no instances of vinyl chloride leaching from gasketed PVC pipes manufactured in North America for water transmission and distribution in sizes 4 to 60 inches. According to a 2002 EPA study on permeation and leaching, some ungasketed (solvent cemented) PVC pipes less than 2 inches in diameter manufactured in the U.S. prior to 1977 experienced vinyl chloride leaching. However, no instances of vinyl chloride

			PVC drinking water pipes manufactured in Saudi Arabia and aged 27 months (Table 4). . . . Decreasing pH was associated with an increase in migration for all five metals (barium cadmium, calcium, lead, tin) studied. Elevating the water temperature from 35◦C to 45◦C increased tin, barium, and calcium concentrations but had no effect on water levels of lead or cadmium. A positive association for all metals was also seen with an increase in total dissolved solids (TDS).	leaching from any North American PVC pipe manufactured post-1977 have ever been cited").
100	§ I.B.2.c., p. 14 FN 134 – Zhang (2014)		*Parenthetical:* (reporting that "the main leachates from PVC pipe are metal stabilizers like lead, tin, barium, [and] calcium . . . , vinyl chloride monomers . . . and other contaminants related with plasticizers, antioxidants and lubricants commonly used in pipe manufacturing processes") At present, a number of papers have been published focusing on the contaminant migration from PVC and PE pipes, with some effort toward PP materials. Extensive research has been carried out to show that the main leachates from PVC pipe are metal stabilizers like lead, tin, barium, calcium and etc. (Adams et al., 2011, Al-Malack, 2001, Lasheen et al., 2008), vinyl chloride monomers (Al-Malack, 2004, Al-Malack and Sheikheldin, 2001, Al-Malack et al., 2000, Walter et al., 2011) and other contaminants related with plasticizers, antioxidants and lubricants commonly used in pipe manufacturing processes (Heim and Dietrich, 2007, Löschner et al., 2011, Skjevrak et al., 2003)	Again, this study relies on research conducted in various foreign countries and on foreign-produced materials to support its statements regarding leachate. Differences in regional production standards and practices must be taken into account. For example, the use of lead stabilizers in PVC pipe was common in the EU and other regions, but that was not the case in the United States.
101	§ I.B.2.c., p. 14 FN 134 – Lasheen (2008)		Lead release from PVC pipes showed to be the highest of all pipes. Results clearly demonstrate the steady increase in lead concentration released from the PVC pipes with respect to time. Concentrations were higher than the lead standard in drinking	This study assesses the release of substances from PVC, PP, and GI pipes used in Egypt that are not subject to U.S. standards and regulations. Lead stabilizer was never used in PVC pipe in the U.S., nor is it currently permitted under the NSF 61 standard.

			water adopted in Egypt. A study carried out by Al-Malack [14] showed by using a circulatory method that lead concentration migrated from PVC pipes into the circulated water after 10h of exposure reached a value of 0.43 mg/l and by the end of the experiment (48 h), it increased to 0.78 mg/l.	
102	§ I.B.2.c., p. 15 FN 135 – Walter (2011)	The rate of accumulation varies significantly according to the conditions of use, as well as the age, origin and manufacturer of the pipe.	Although all values measured in this study are below the EPA's MCL of 2.0 mg/L, many readings, especially at longer times, were quantifiable and, therefore, above the MCL-Goal of 0 mg/L. For new pipe manufactured in the US, long term studies in the lab suggested equilibrium concentrations of about 300 ng/L. In time frames that simulate overnight stagnancy in a home (5e8 h), VC levels varied from below detection (<6 ng/L) to 20 ng/L, with pipe age and/or manufacturer having some effect. Viewing our results in the context of other studies, considerable differences in VC accumulation can occur across different manufacturers, especially pipe manufactured outside the U.S. and before 1977.	Although we do not contest that the age, origin, and manufacture of a pipe may impact its performance, the referenced study of PVC/CPVC pipe demonstrated that the material did not exceed EPA's MCL under numerous tested and field conditions. Refer to discussion at line 98 for a more detailed discussion.
103	§ I.B.2.c., p. 15 FN 136 – Stern (2008)	For example, "early era" pipes, which were built before 1977 and remain in use today, may be especially likely to release dangerous quantities of vinyl chloride.	"Early-era" [manufactured prior to 1977] PVC potable water pipes tended to leach vinyl chloride monomer (VCM) into drinking water. For example, the Kansas Department of Health and Environment (KDHE 1998) conducted a systematic survey of 125 rural drinking water distribution systems using PVC pipe manufactured before 1977. Sampling was conducted between June and October (the months expected to have the warmest water temperatures). The results showed that almost 10 percent of the systems had levels of vinyl chloride exceeding the USEPA action level of 2 ppb (KDHE 1998) and attributed to leaching.	As discussed above, vinyl chloride leaching prior to 1977 was limited to small diameter pipe (less than 2 inches). Since these small diameter pipes were not widely used at the time, they are even less prevalent today. Carroll, Jr, William & Eckstein, Dave, *Vinyl chloride in rural water from pre-1977 applications of PVC pipe.* Journal of Vinyl and Additive Technology. Vol. 7 (2001) p. 49, Table 1; *see also* Sustainable Solutions Coalition *Life Cycle Assessment of PVC Water and Sewer Pipe and Comparative Sustainability Analysis of Pipe Materials (2018), https://www.uni-bell.org/files/Reports/Life_ Cycle_Assessment_of_PVC_Water_and_Sewer _Pipe_* and_Comparative_Sustainability_Analysis_of_Pipe _Materials.pdf (stating that, "EPA has found no instances of vinyl chloride leaching from gasketed PVC pipes manufactured in North America for water transmission and distribution in sizes 4 to 60 inches. According to a 2002 EPA study on permeation and leaching, some ungasketed (solvent cemented) PVC pipes less than 2 inches in diameter

				manufactured in the U.S. prior to 1977 experienced vinyl chloride leaching. However, no instances of vinyl chloride leaching from any North American PVC pipe manufactured post-1977 have ever been cited").
104	§ I.B.2.c., p. 15 FN 138 – Heudorf (2007)	phthalate plasticizers are not chemically bound to the plastic matrix and thus "leach, migrate or evaporate" into surrounding media and the atmosphere even under ideal conditions,	This paper presents an overview on current risk assessments done by expert panels as well as on exposure assessment data, based on ambient and on current human biomonitoring results. As the phthalate plasticizers are not chemically bound to PVC, they can leach, migrate or evaporate into indoor air and atmosphere, foodstuff, other materials, etc. Consumer products containing phthalates can result in human exposure through direct contact and use, indirectly through leaching into other products, or general environmental contamination. Humans are exposed through ingestion, inhalation, and dermal exposure during their whole lifetime, including intrauterine development.	This is true and not in dispute, but the potential for migration is exaggerated or mischaracterized. The article fails to draw clear and consistent conclusions between exposure and the use of PVC products.
105	§ I.B.2.c., p. 15 FN 138 – Shashoua (2003)		*Parenthetical:* (reporting that "[i]n many international museum collections, degradation of plasticized PVC materials . . . has been detected as early as 5 years after acquisition," and concluding that "[t]he rate and extent of deterioration of plasticized PVC and the migration and loss of DEHP plasticizer [are] related"). Model PVC sheets and naturally aged material, plasticized with DEHP, were exposed to various environments under accelerated thermal ageing. Non-destructive examination techniques were used to identify and quantify changes in visual, chemical and structural properties of samples during thermal ageing (Table 1). Weight loss was used to quantify loss of plasticizer. Attenuated total reflection Fourier transform infrared (ATR-FTIR) spectroscopy was used to quantify con-	The age of the museum collection items is unknown and do not likely represent performance of products produced in the last 40 years.

			centration of DEHP at surfaces. Optical densitometry was used to quantify darkening of samples. Sheets were prepared from a PVC plastisol, prepared by Hydro Polymers in Norway, comprising a liquid dispersion of medium molecular weight PVC polymer, DEHP plasticizer and barium/zinc laurate as thermal inhibitor. Chemical composition of the plastisol was characterized by thin-layer chromatography of an ethe-real extract. To produce model sheets representing the range of plasticizer concentrations found in museum collections, the plastisol was diluted using DEHP, resulting in plasticizer concentrations of 33.3, 37.5, 44.4 and 50.0% by weight. Pigments were omitted since they were likely to confuse perception of discolouration by the PVC component. Plastisols were heat pressed at 180C for 90 s, achieving complete fusion between PVC and DEHP with minimal yellowing and without solvent residues. Sheets 0.5 mm thick were produced and conditioned for 14 days under ambient condition (ASTMD2115-671980). Rectangular-shaped samples weighing 1g (approximately 50x30 mm) were cut from the sheets for exposure to various environments.	
106	§ I.B.2.c., p. 15 FN 139 – Fossi (2012)	resulting in pervasive contamination.	*Parenthetical:* ("[Phthalates] are not covalently bound to plastic and migrate from the products to the environment, thus becoming ubiquitous contaminants.") Moreover, contaminants such as phthalates and polycyclic aromatic hydro- carbons (PAHs) are among the principal constituents of plastics. The dialkyl or alkyl/aryl esters of 1,2-benzenedicarboxylic acid, commonly known as phthalates, are high-production-volume synthetic chemicals; moreover, they are not	Addressed in main comments and assessing bond strength of plasticizers to polymeric matrix.

			covalently bound to plastic and migrate from the products to the environment, thus becoming ubiquitous contaminants (Latini et al., 2009). Latini et al. 2009: "By means of a LC/LC-MS/MS method we tested the presence of several different phthalate metabolites in breast milk from 62 healthy mothers living in Southern Italy. . . These findings indicate that exposure to phthalates through breast milk in Southern Italian infants is comparable to that of other countries, thus confirming that human milk may represent an additional potential source of phthalate exposure in a population at increased risk. However, different milk concentrations of MiBP may suggest a different pattern of usage of di-iso-butyl phthalate in Europe, as compared to USA, whereas for the first time, we detected an oxidative DiNP metabolite, whose significance remains unclear."	
107	§ I.B.2.c., p. 15 FN 139 – Kaplan (2013)		*Parenthetical:* (reporting that "[p]hthalates have been detected in all aspects of the environmental [sic]: water, air, sediment, biota, marine, and freshwater ecosystems") Benzyl butyl phthalate (BBP) is a phthalic acid ester used to make plastic products such as toys, packaging materials, and vinyl flooring more pliable and durable (Wibe et al., 2002). Since phthalate esters are not chemically bonded within the polymer matrix, they may migrate from the plastic composite into the environment (Adams et al., 1995). Phthalates have been detected in all aspects of the environmental: water, air, sediment, biota, marine, and freshwater ecosystems (Fatoki and Vernon, 1990; Giam et al., 1978). Since low-level environmental contamination may disrupt shoaling and	

			negatively impact both fitness and transmission of social information (Reader et al., 2003), the focus of this study was to examine the influence of BBP on shoaling behavior in F. heteroclitus to determine if the model (fish and behavior) can be utilized as a bioindicator of sub-lethal BBP exposure.	
108	§ I.B.2.c., p. 15 FN 139 – Abdel daiem (2012)		*Parenthetical:* (explaining that "the slow release of phthalates from plastics and other phthalate containing materials due to weathering" accounts for much of the presence of these compounds in the environment). The major portion of phthalic acid esters found in the environment is as a result of the slow release of phthalates from plastics and other phthalate containing materials due to weathering. These pollutants are refractory to the environmental microorganisms, and their accumulation in natural waters causes their wide distribution within aqueous systems like rivers, lakes, and ground waters, as well as a noticeable influence on the ecological environment. Phthalates are not chemically bound to polymer matrixes and can be readily dispersed into the environment during their production and use and after their disposal. Phthalate plasticizers have become ubiquitous contaminants because they are not covalently bound to PVC and can leach, migrate or evaporate into foodstuffs or any other material, indoor air and the atmosphere (Pant et al., 2008). Because of their low solubility, phthalates tend to be concentrated from wastewater into sewage sludge, which is then used as a soil amendment, implying the exposure of soil microbial communities, plants, and animals	

			to these compounds and their introduction into the food chain.	
109	§ I.B.2.c., p. 15 FN 140 – Abdel daiem (2012)	High concentrations of these compounds seep into soil and groundwater following conventional landfill disposal	In landfills, the leachate is formed from refractory organic contents during biological degradation. The leachate has high biochemical oxygen (BOD) and chemical oxygen (COD) demands and passes down through the layers of the landfill through the action of rainwater, thereby potentially contaminating the soil and drinking waters (Kuch and Ballschmiter, 2001). Analyses of landfills have revealed alkylphenols (APs), Bisphenol A (BPA), phthalic acid esters (PAEs), organotin compounds (OTs), styrene and estradiol (Asakura et al., 2004).	The article raises questions on whether the landfill is being properly maintained. However, the studies that this article points to regarding the analysis of landfill leachate and the measurement of phthalates in water and sediment are based on observations in various parts of Europe and Asia. Accordingly, the questions raised by this article may not be representative of U.S. conditions.
110	§ I.B.2.c., p. 15 FN 140 – Chatterjee (2010)		*Parenthetical:* ("Appreciable amounts of phthalates have been detected in liquid samples withdrawn from landfills and in landfill leachates.") Phthalates are weakly bound to plastics and therefore readily leach from plastic products into the environment. Appreciable amounts of phthalates have been detected in liquid samples withdrawn from landfills (Oman and Hynning 1993) and in landfill leachates (Bauer et al. 1998; Jonsson et al. 2003a).	The evidence offered is supported by European landfill studies. This is not reflective of conditions in the U.S.
111	§ I.B.2.c., p. 15 FN 140 – Teuten (2009)		*Parenthetical:* (reporting that discarded PVC and other plastics waste release phthalate plasticizers "after their disposal, for example in landfills") The objective of this paper is to review the phenomena by which plastics released to the environment serve as carriers of organic contaminants to wildlife. . . Section 2 reviews the migration and degradation of plasticizers (phthalates), additives (organotin compounds and nonylphenols (NP)) and monomers (BPA), while §3 focuses on landfill leachate as a source of	The cited article is a literature review and does not offer direct citations for its statements regarding landfills. The article compares the migration of DEHP (one of the phthalates cited by CBD in the petition) with DMP and states that DEHP, like other high-molecular weight phthalates, are actually more resistant to migration compared to DMP.

			plastics-derived endocrine-disrupting compounds The release may take place during the service life of the plastics or after their disposal, for example in landfills. Both the landfill compartment and other potential receptors such as sediments represent complex environments with multiple chemical and biological processes occurring concurrently. In landfills, plastics are exposed to an extraction solvent in the form of acidic (pH 5 – 6) leachates with high ionic strength and neutral or alkaline leachates containing high-molecular-weight organic compounds. The different leachates have not only different potentials to extract and transport, but also different biological populations with the potential to degrade or transform the released additives. Plasticizers, which are the largest group of additives in polymers, range from molecular weights of approximately 200 to almost 700 g mol – 1 and cover water solubility from g l – 1 to sub-mg l – 1. Phthalates (or more chemically correct, alkyl/aryl esters of 1,2-benze- nedicarboxylic acid) are the most common plasticizers and may account for more than 60 per cent of polymers of PVC (Giam et al. 1984). Dimethyl phthalate (DMP) is fairly easily released from its resin, as soon as the DMP-containing product is landfilled, owing to its relatively high water solubility, i.e. there is a continuous depletion of DMP from the resin surface, and the negative concentration gradient from the inside to the surface causes the migration. In contrast, the higher-molecular-weight phthalates, such as diethyl- hexyl phthalate (DEHP), are more	

			resistant to migration owing to their hydrophobicity, which causes less release from the polymer surface to leachate compared with DMP.	
112	§ I.B.2.c., p. 15 FN 141 – Depledge (2013)	ultimately collecting in aquatic ecosystems and entering marine food webs.	On the 5th and 6th June 2013, ca. 100 scientists from 6 countries met for a workshop at the University of Siena, Italy, to review current knowledge and to clarify what is known, and what remains to be investigated, concerning plastic litter in the sea. Several key facts were highlighted at the workshop: 1) Plastic litter is diverse and now very widely distributed in the marine environment. 2) Many kinds of plastic litter are extremely persistent, often for several decades. 3) Some kinds of marine organisms are particularly vulnerable to plastic litter, including turtles, marine mammals, suspension feeders and deposit feeders. 4) Some plastics components and their constituent chemicals can be transferred through marine food webs (eg. phthalates).	This article reports a number of statements from the June 2013 workshop at the University of Siena (Italy) that were relevant to policymakers and scientists and for which there was agreement. The workshop was designed to contribute further to the European Marine Strategy Framework Directive (MSDF).
113	§ I.B.2.c., p. 15 FN 141 – Cheng (2013)		*Parenthetical:* ("Human exposure to phthalate ester mainly occurs through dietary intake, due [in part] to the bioaccumulation of phthalate esters in food chains.") There is no chemical bond between the phthalate esters and plastics in their mixture, therefore phthalate esters are easily released into the environment via direct release, leaching, evaporation, abrasion, and migration (Staples et al., 1997; Wittassek et al., 2011). Recent studies showed that in China, phthalate ester pollution is widespread in the environment, with relatively high concentrations observed in	The major objectives of this study were: (1) to analyze the bioaccessibility of phthalate esters contained in fish muscle and, (2) to assess potential health risks based on raw and digestible phthalate ester concentration in fish muscle. The fish used for the study were purchased from Hong Kong markets. Accordingly, the study is not directly relevant to exposure in the U.S.

			Guangdong province and northeast China (Chen et al., 2012). In addition, DEHP was the main phthalate in environmental media (air, water, sediment and soil) (Chen et al., 2012; Huang et al., 2008). Human exposure to phthalate ester mainly occurs through dietary intake, due to the bioaccumulation of phthalate esters in food chains and the frequent use or unintended presence of phthalates in various food materials during processing, storing, and transport (Staples et al., 1997; USEPA, 2007). Relatively high concentrations of phthalate esters in foodstuffs (e.g.: aquatic product 10.6–52.41 mg/kg, vegetable 0.43–6.85 mg/kg, grain 5.79–39.94 mg/kg) (Chen et al., 2012; Mo et al., 2009) and subsequently high daily intake level of phthalate esters (128.6 μg/kg bw/day) were observed in Pearl River Delta (PRD), China (Chen et al., 2012). It was revealed that concentrations of phthalate esters in human body were positively correlated with fish consumption, which dominated the total dietary phthalate ester intake (Chen et al., 2012). Aquatic products are a major dietary source of protein for most of Hong Kong residents. Most of the aquatic products available in Hong Kong markets mainly come from the surrounding coastal and fresh water fish farms in PRD. The Taiwan phthalate incident in 2011 (Li and Ko, 2012) has rendered phthalate esters a general public health concern.	
114	§ I.B.2.c., p. 15-16 FN 142 – Jobling (1995)	As a result of their widespread use and significant tendency to migrate, phthalates are the most abundant anthropogenic chemicals in the environment.	In contrast, phthalates are the most abundant man-made chemicals in the environment (29). They are produced industrially in large quantities, mainly to impart flexibility into plastics, and can leach out of these materials into water, soil, or food overtime. BBP is also used in the production of vinyl floor tiles, adhesives, and synthetic leather; DBP is more common as a plasticizer in food-packaging materials, PVC, the cellulosic, and certain types of	Many of the phthalates mentioned in the Jobling article are no longer used in PVC. Indeed, the Flexible Vinyl Alliance submitted a petition to the Food and Drug Administration in 2018 to amend the food additive regulations to remove 26 orthophthalates, including BBP, DBP, dimethyl phthalate, diethyl phthalate, and DnOP, among others, in various food-contact applications because these uses have been permanently abandoned. *Flexible Vinyl Alliance; Filing of Food Additive Petition*, 83 Fed. Reg. 56,750 (Nov. 14, 2018).

			elastomers (30-32). Thousands of tons of plastics are disposed of annually in landfill sites, thus enabling phthalate esters to migrate into groundwaters via the soil. The ubiquity of these compounds in the aqueous environment is well known, and their presence is reported in river, waste, and drinking waters as well as in fish and sediments (33-39). Commonly detected species include DBP, dimethyl phthalate, diethyl phthalate, DEHP, di-n-octylphthalate, BBP, and DEHA (16).	
§ I.B.2.d. Persistence				
115	§ I.B.2.d., p. 16 FN 143 – Adbel daiem (2012)	Multiple researchers have acknowledged the persistent nature of phthalate plasticizers	*Parenthetical:* (listing phthalates among "persistent toxic organic compounds") This article describes the most recent methods developed to remove phthalic acid esters (PAEs) from water, wastewater, sludge, and soil. It is common practice to dispose of biosolids by treating the sludge and using it for soil conditioning (biosolids represent half of European sludge production), and the resulting accumulation of persistent toxic organic compounds, as PAEs, in the soil poses a growing threat to ecosystems and human health. The use of sewage sludge in agriculture can produce human exposure during its application or through the resulting introduction of these compounds into the food chain. There is an urgent need to ensure that sludge is free from these contaminants before its utilization (Amir et al., 2005).	The pincite that CBD uses (and references in their parenthetical) refers only to a passing description of PAE's as "persistent toxic organic compounds." The article does not discuss the persistence of phthalates in detail.
116	§ I.B.2.d., p. 16 FN 143 – Klopffer (1996)		*Parenthetical:* (arguing that DEHP "should be considered as a persistent chemical," because "it is not degraded in anaerobic media") The first specialized paper deals with chemicals in sewage sludge, produced in or	This article discusses the nature and properties of DEHP and not potential exposure or effects from discarded PVC products.

			passing through sewage treatment plants. The sewage treatment plants are, so to speak, the "frontier guards" of the technosphere where noxious or undesirable components of the sewage are to be retained and decomposed: Substances not retained here get out of the control by man and thus enter into the environment (defined as the opposite of the technosphere (1,5]). DEHP is said to be aerobically biodegradable, but nevertheless it is present in sewage sludges and sediments [28] nearly ubiquitously. Since it is not degraded in anaerobic media (as typical sediments are), DEHP can accumulate despite its (aerobic) degradability and should be considered as a persistent chemical [2,19]. In the tcchnosphere [1], a huge "depot" (millions of tons worldwide 15J) of DEHP has been building up, mainly contained in soft PVC. Hence, a slow leaking of DEHP into the water-path will continue and increase with time.	
117	§ I.B.2.d., p. 16 FN 143 – Jobling (1995)		*Parenthetical:* (describing phthalate plasticizers as "environmentally persistent chemicals") The study tested several "environmentally persistent chemicals" found in sewage effluents to determine their estrogenic activity. As relevant to the petition, this included phthalate plasticizers DEHP, BBP, and DBP. In conclusion, we have discovered that a surprisingly large proportion of environmentally persistent chemicals are weakly estrogenic . . . Although the chemicals we tested possess some common structural features (such as a benzene ring), there is no obvious part of their molecular structure that might be expected to enable	Although the article discusses the persistence of several phthalates, it is not specific to potential exposure or effects from discarded PVC products.

			binding to the estrogen receptor, and hence one cannot easily deduce which chemicals are and which are not estrogenic.	
118	§ I.B.2.d., p. 16 FN 144 – Mankidy (2013)	and the widespread occurrence of these compounds clearly demonstrates that their massive rate of synthesis outpaces natural removal processes	This study investigated cytotoxicity, endocrine disruption, effects mediated via AhR, lipid peroxidation and effects on expression of enzymes of xenobiotic metabolism caused by di-(2-ethy hexyl) phthalate (DEHP), diethyl phthalate (DEP), dibutyl phthalate (DBP) and benzyl butyl phthalate (BBP) in developing fish embryos. A number of studies label phthalates as innocuous agents that do not persist in the environment (Staples, 1997) and exhibit very little tendency to bioaccumulate (Gobas F 2003). However, it is important to note that the massive rate of synthesis of phthalates could supersede the natural rate of removal, leading to an eventual accumulation of these chemicals in the environment (aquatic sediments, particulate material) and ultimately in humans.	Although the article discusses the persistence of several phthalates, it is not specific to potential exposure or effects from discarded PVC products.
119	§ I.B.2.d., p. 16 FN 145 – Migliarini (2011)	For example, DEHP is abundant in surface waters, despite its medium- specific half-life of fewer than five weeks.	The aim of this review is to summarize the literature from experimental animal studies demonstrating the impairment of body weight raised by the deregulation of peptidergic signals as well as by the activation of key metabolic molecular targets. Among plasticizers, the phthalate di-ethyl-hexyl-phthalate (DEHP) worth particular attention for its wide presence in the soil and water that has been considered a priority environmental concern by North American and Europe governments. When sus- pended in the water, its half life is around 2–5 weeks while, in the sediments, captured by particulate matter, has been estimated to be over 100 years due to unavailability for biodegradation.	According to EPA, DEHP released to water systems will, indeed, biodegrade fairly rapidly (half-life 2-3 weeks). However, the measurements of DEHP in surface water stem from observations made at least a decade ago.

120	§ I.B.2.d., p. 16 FN 145 – Magdouli (2013)		*Parenthetical*: (observing that "[t]he abundance of DEHP in [the] aqueous environment is mainly related to its extensive utilization and production" because, "[d]ue to its highly hydrophobic properties, the principal fate of DEHP in water and wastewater could be adsorption to the suspended solids") While understanding the adverse impacts of DEHP in the environment, this review also highlights various issues concerning its sources, toxicity, distribution, fate and the different treatment technologies applied so far for their removal. The abundance of DEHP in aqueous environment is mainly related to its extensive utilization and production. Due to its highly hydrophobic properties, the principal fate of DEHP in water and wastewater could be adsorption to the suspended solids. According to Khan and Jung (2008), DEHP concentrations are found in surface water within the range of 0.33 $\mu g\ L^{-1}$–97.87 $\mu g\ L^{-1}$. For instance, the concentration of DEHP reported by Yuwatini et al. (2006) in river water was between 8 $\mu g\ L^{-1}$ and 25 $\mu g\ L^{-1}$. In Germany (0.05-0.06 $\mu g\ L^{-1}$), Greece (0.93 $\mu g\ L^{-1}$) and Croatia (0.247 $\mu g\ L^{-1}$), various DEHP concentrations in the micron range were detected in drinking water (Luks-Betlej et al., 2001; Psillakis and Kalogerakis, 2003; Huerta-Fontela and Ventura, 2008). In aquatic environment, the half-life of DEHP under sunlight irradiation was 390 days in acidic conditions and reached 1600 days in neutral medium (Lertsirisopon et al., 2009). Under aerobic conditions, more than one week or month is required for DEHP	

			biodegradation, whereas in anaerobic conditions, the half-life of DEHP exceeds 1 month (Lertsirisopon et al., 2009).	
121	§ I.B.2.d., p. 16 FN 146 – Migliarini (2011)	Further, as a consequence of its "highly hydrophobic" character, this compound also tends to form strong bonds with suspended particulates and ultimately accumulates in aquatic sediments, where estimates indicate it will persist for more than 100 years.	The aim of this review is to summarize the literature from experimental animal studies demonstrating the impairment of body weight raised by the deregulation of peptidergic signals as well as by the activation of key metabolic molecular targets. Among plasticizers, the phthalate di-ethyl-hexyl-phthalate (DEHP) worth particular attention for its wide presence in the soil and water that has been considered a priority environmental concern by North American and Europe governments. When suspended in the water, its half life is around 2–5 weeks while, in the sediments, captured by particulate matter, has been estimated to be over 100 years due to unavailability for biodegradation.	
		§ I.B.2.e. Degradation Potential and Rate of Degradation		
122	§ I.B.2.e., p. 16-7 FN 148 – Wams (1987)	Once released from discarded PVC, phthalates resist physical and chemical degradation.	In this paper [ ] applications, emissions, environmental fate, human toxicity and exposure, ecotoxicology and possibilities for emission-reduction [of DEHP] are discussed. DEHP is emitted to the environment during the production of plastics and plastic products, during their use and after disposal. In the environment, physico-chemical degradation of DEHP is practically non-existent. Biodegradation occurs readily under aerobic conditions (t~ = 2 4 weeks), but not under anaerobic conditions.	Phthalates refer to a family of substances with different characteristics. The referenced study only addresses DEHP, which is too narrow to support CBD's broad statement regarding phthalate plasticizers as a class.
123	§ I.B.2.e., p. 17 FN 148 – Magdouli (2013)		*Parenthetical:* (reporting that the half-life of DEHP under sunlight irradiation in aquatic environments may exceed four years)	Again, this study only addresses DEHP, which is too narrow to support CBD's broad statement regarding phthalate plasticizers as a class. In addition, the half-life cited by CBD represents a 'worst-case' degradation scenario for DEHP. Indeed, prior citations

			While understanding the adverse impacts of DEHP in the environment, this review also highlights various issues concerning its sources, toxicity, distribution, fate and the different treatment technologies applied so far for their removal. In aquatic environment, the half-life of DEHP under sunlight irradiation was 390 days in acidic conditions and reached 1600 days in neutral medium (Lertsirisopon et al., 2009).	offered by CBD cite the half-life of DEHP as less than five weeks in aquatic environments.
124	§ I.B.2.e., p. 17 FN 149 – Wams (1987)	Scientists have long recognized that the biological transformation of phthalate plasticizers "comes to a standstill" under anaerobic conditions, such as those found in deep soils, aquatic sediments, and most landfills.	In soil, biodegradation proceeds as in surface waters [34]. In the upper layer, where aerobic conditions exist, the biological transformation of DEHP occurs quite easily. Deeper down, where anaerobic conditions are predominant, degradation comes to a standstill. Under anaerobic conditions the degradation rate of DEHP is reduced to zero. Other phthalic esters may still be degraded under these conditions, but at a much reduced rate [31, 32].	While DEHP does partition to soils and sediments due to its extremely low water solubility, it is still subject to degradation and even microbial decomposition depending on the temperature and microbes present. *See* Zhu F, Zhu C, Zhou D, Gao J. *Fate of di (2-ethylhexyl) phthalate and its impact on soil bacterial community under aerobic and anaerobic conditions.* Chemosphere. 2019 Feb;216:84-93. doi: 10.1016/j.chemosphere.2018.10.078. Epub 2018 Oct 18. PMID: 30359920; *see also* ATSDR Toxicological Profile: DI(2-ETHYLHEXYL)PHTHALATE, https://www.atsdr.cdc.gov/ToxProfiles/tp9-c5.pdf .
125	§ I.B.2.e., p. 17 FN 150 – Horn (2004)	In aerobic environments, the partial degradation of these chemicals yields metabolites that are more harmful than the original plasticizers, including 2-ethylhexanoic acid, 2-ethylhexanal and 2-ethylhexanol	Because millions of tonnes of plasticizers are incorporated into plastics every year and are ultimately released to the environment, the objective of this work was to establish whether the toxic metabolites are also observed in the environment. Plasticizers such as DEHP or DEHA can be degraded by a common soil bacterium in the presence of an easily used carbon source (Nallietal.,2003). The degradation was not complete and resulted in the production of metabolites including 2-ethylhexanol, 2-ethylhexanoic acid and monoesters such as 2-ethylhexyl phthalate. A proposed pathway for the degradation of DEHP or DEHA is shown in Fig. 1. The final metabolite for both DEHP and DEHA was 2-ethylhexanoic acid, which was observed to	The origin of the metabolites is unknown.

			be particularly resistant to further degradation. It must be acknowledged that there are other possible sources of the alcohol and acid besides the metabolism of plasticizers. For example, they could originate from plasticizer production processes, the use of lubricants and surfactants, as well as the pharmaceutical industry (Staples,2001). However, it has been claimed that these sources are well contained and that the main sources of exposure would be due to accidental spills (Staples, 2001). These sources could not account for the quantities observed in our studies nor their presence in many different environmental samples. In addition, the mono-ester observed in the sediment sample could only come from the metabolism of the di-ester, since the half-life of DEHP degradation by abiotic alkaline hydrolysis has been reported to exceed 1000 years at neutral pH (Wolfeetal.,1980). Thus, these observations support the argument that the metabolites that were observed in the environment originated from the biodegradation of plasticizers. As plasticizers themselves are ubiquitous and can be partially degraded by common soil bacteria, we conclude that plasticizer metabolites will also be widespread. While the levels of metabolites observed in this study may not result in acute aquatic toxicity, it is likely that the intractability will lead to a tendency for them to persist in the environment.	
126	§ I.B.2.e., p. 17 FN 150 – Nalli (2006)		The objective of the present study is focused on determining the fate of the 2-ethylhexanol component during the biodegradation of DEHA or DEHP by a single bacterium by tracking its accumulation, bioconversion and	The environmental fate of DEHP and its metabolites are very sensitive to the conditions of exposure. Certain microbes attack these molecules more than others, the pH of the soil or water is important, as is the temperature. So, to definitively state a result using a single bacterium only produces a single data point of biologic digestion where dozens of important

			volatilization. This will contribute to the understanding of this neglected aspect of the degradation mechanism of DEHP and DEHA by a common soil micro-organism. The work presented here demonstrated that another metabolite in addition to the previously identified 2-ethylhexanol and 2-ethylhexanoic acid (Nalli et al., 2002) will contribute to this overall environmental problem. Analyses of the gas phase has shown that significant quantities of 2-ethylhexanal were produced from the degradation of plasticizers, even though it had not been observed in previous studies in which only the aqueous phase was considered.	microbes are available for attacking these substances. In the real world, some of the metabolites of DEHP and DEHA could be digested by microbes not tested in this study. *See* Fengxiao Zhu *et al., Fate of di (2-ethylhexyl) phthalate and its impact on soil bacterial community under aerobic and anaerobic conditions,* CHEMOSPHERE 216:84-93 (Feb. 2019) *doi: 10.1016/j.chemosphere.2018.10.078.*; *see also* ATSDR *Toxicological Profile: di(2-ethylhexyl)phthalate,* https://www.atsdr.cdc.gov/ToxProfiles/tp9-c5.pdf (stating that "[a]naerobic biodegradation of DEHP in sediments was reported to occur, but more slowly than under aerobic conditions").
127	§ I.B.2.e., p. 17 FN 151 – Horn (2004)	Field studies have detected these acutely toxic metabolites in surface waters, river sediment, freshly fallen snow and even tap water, giving rise to significant concern about potential consequences for human health and the environment.	Because millions of tonnes of plasticizers are incorporated into plastics every year and are ultimately released to the environment, the objective of this work was to establish whether the toxic metabolites are also observed in the environment. Most of the environmental samples also contained one or more of the metabolites, which is consistent with recent studies of the degradation of these plasticizers using pure cultures (Nalli et al., 2003). This work had shown that 2-ethylhexanoic acid was a very intractable metabolite and, thus, it was not surprising to find appreciable amounts of it in the environment . . . River sediments had the highest concentrations of the alcohol and acid. This is consistent with the fact that these compounds are relatively hydrophobic and thus likely to partition into the sediments . . . The samples of snow and tap water were expected to have the lowest concentrations of the compounds of interest. In fact, even these were contaminated. The data for the sample of snow revealed that precipitation can act as a means of introducing plasticizers into surface waters .	The citation offered to support the detection of DEHP metabolites in field studies only sampled from Canadian locations.

			. . The tap water contained small amounts of the plasticizers and the acid metabolite. It is probable that this metabolite originated from the degradation of the plasticizers, but it is unknown whether this compound entered the water during processing or distribution or if it came from the water source (St. Lawrence River) and passed through the treatment system.	
128	§ I.B.2.e., p. 17 FN 151 – Barnabé (2008)		The presence of bis (2-ethylhexyl) ester plasticizers (BEHP, BEHTP, BEHA) and their metabolic intermediates was investigated in the process streams and residues of a sewage treatment plant in Montreal, Canada. This treatment facility incorporates primary and physicochemical treatment steps and serves a large urban population. With a maximal capacity of about 7.6 million m3 per day and serving over 1 880 000 citizens, it is the largest primary–physicochemical sewage treatment plant in North America (City of Montreal, 2006). The study involved the quantification of plasticizers and their breakdown products entering the treatment plant in the influent sewage and estimation of the amounts ultimately released in treated effluents and process residues. This study demonstrated that there were significant quantities of BEHA, BEHP and BEHTP in the influents, effluents and process streams of a large urban physicochemical sewage treatment plant. Furthermore, all of the expected biodegradation products, 2-ethylhexanol, 2-ethylhexanal and 2-ethyl- hexanoic acid, were found in all of the process streams.	The metabolites identified in this study are the product of the degradation of BEHP, BEHTP, and BEHA. None of these phthalates are identified in the CBD petition.
	§ I.B.2.f. Bioaccumulation			
129	§ I.B.2.f, p. 17 FN 152 – Wang (2013)	Fish and other aquatic organisms accumulate phthalates directly from the environment and as a	This study quantified the aqueous uptake and dietary assimilation (trophic transfer) of two endocrine disrupting compounds (dioxin and phthalic acid) in green mussels,	This is a laboratory study of the uptake of dioxins and DBP from water and not potential exposure or effects from discarded PVC products. To that end, the experimental seawater and green mussels were collected from Hong Kong

		consequence of ingesting contaminated food and particles.	and found that the aqueous uptake was a predominant route for the bioaccumulation of these two compounds in the mussels. The study "examined a model hexachlorobenzo-p-dioxin and a model dibutyl phthalate using radiotracer methodology, which can provide an accurate approach in quantifying the biokinetics of these compounds in aquatic organisms. We finally used a kinetic equation to predict the route of these two contaminants accumulation in the mussels." The dibutyl phthalate [ring-14C(U)] (>99% purity, specific activity of 60 mCi $mmol^{-1}$, dissolved in ethanol, molecular weight 378.34) was purchased from American Radiolabeled Chemicals, Ltd.	and thus, are not necessarily reflective of, or relevant to, the U.S. environment.
130	§ I.B.2.f, p. 17 FN 153 – Bhattacharya (2010)	For example, scientific evidence indicates that certain microscopic plastic fragments enter the marine food web by adhering to algae.	The study found that adsorption of nanosized plastic beads has been found to hinder algal photosynthetic activities and promote their relative oxygen species production. Such adsorption depended on the physico-chemical properties of the plastic and the morphological and biochemical properties of the algae. Positively (Amidine Latex) and negatively (Carboxyl Latex) charged polystyrene (PS) beads were purchased from Molecular Probes. The primary size and surface area of both types of beads were 20 nm and 2.5 × 106 cm2/g, respectively. The amidine nanoparticles were stable at low pH and hence were dispersed in MES buffer with a pH of 6 at room temperature. The carboxyl nanoparticles were stable at neutral to high pH and were dispersed with Milli-Q water with a pH of7.4 at room temperature. The PS nanoparticles existed as small agglomerates in both dispersions, and a complete breakdown by sonication was not performed, since this would not replicate a	The study only focuses on polystyrene bead adsorption by algae; no discussion of plastic moving throughout the food web other than to mention that algae are at the bottom of the food chain and algal photosynthesis is a major source of earth's oxygen. The study is conducted in a lab environment and there is no evidence to show that the plastic fragments are in fact adhering to algae naturally in the environment, nor are the fragments shown to enter the food web outside of the lab environment; only that algae has the capability to bind to the plastic. There is also no reference to PVC and no connection between PS beads and PVC.

			realistic representation of the natural conditions. AUV-vis spectrophotometer (Biomate 3) was used to quantify the amount of the PS beads adsorbed on the algae. The Freundlich model was used to fit the adsorption isotherms for PS beads. . . These measurements suggested that positively charged PS beads possessed a higher binding affinity than the negatively charged ones for the algae . . . Such consistency implied that cellulose played an essential role in initiating the binding between the algae and the plastics.	
131	§ I.B.2.f, p. 17 FN 154 – Ivar do Sul (2013)	In addition, studies have documented plastic consumption among vertebrates and invertebrates from every feeding guild,	The ingestion of microplastics has been documented for vertebrates (Moore, 2008; Boerger et al., 2010) and invertebrates (Wright et al., 2013) from every level of the marine food web and is very likely to be related to the plastics' size, shape and colour, although additional studies are needed to clarify these questions.	No reference to PVC. This study is limited to microplastics in Archipelago off the coast of Brazil, which were identified as weathered fragments of larger plastic items, most likely polypropylene and polyethylene, only because these are the majority of the plastic polymers produced (no actual testing of composition was done). The study itself does not document the ingestion of plastic consumption among vertebrates and invertebrates, rather, it documents the presence of synthetic polymers in subsurface plankton samples.
132	I.B.2.f, p. 17 FN 155 – Cole (2013)	. . . ranging from zooplankton	Laboratory experiments, in which latex beads were used to model algal ingestion, have shown that zooplankton have the potential to ingest small plastics. Results provide that the majority of zooplankton (13 of 15) exposed to polystyrene beads (7.3−30.6 μm) demonstrated the capacity to ingest microplastics. To ascertain whether zooplankton ingest microplastics we conducted exposures using fluorescent polystyrene beads, and used microscopy to assess uptake. Ingestion was ascertained by viewing specimens at ×40−400 magnification with an Olympus IMT2 inverted light microscope with fluorescence to determine the presence of polystyrene beads (fluorescing yellow-green) within the alimentary canal or body cavity of the zooplankton.	Zooplankton and seawater were collected from the English Channel, UK and exposed to commercial fluorescent polystyrene beads to assess whether they would ingest microplastics. No reference to PVC and no connection between polystyrene beads and PVC.

133	I.B.2.f, p. 17 FN 155 – Setälä (2014)	. . ranging from zooplankton	We showed that plastic micro-spheres were widely ingested by various planktonic taxa in the Baltic Sea. The study "focused on the issue of potential threats of microplastics by carrying out simple grazing experiments with fluorescent micro-spheres and zooplankton. At this point, no attempt to measure ingestion rates was done, just to verify ingestion. In our study we tested experimentally the potential of different Baltic Sea zooplankton organisms to ingest microplastics, to assess the potential of microplastics to enter the planktonic food web." Zooplankton was collected off the coast of Finland. "Fluorescent 10 mm polystyrene spheres (Polysciences inc.) were used at three different target concentrations (A ¼ 1000, B ¼ 2000 and C ¼ 10 000 particles mL^{-1}). We chose 10 mm microspheres because this size of prey is suitable for several zooplankton taxa from protists to copepods." "The study of microsphere passage through the copepods (Eurytemora affinis adults) was done by incubating the animals as described, and then narcotizing the collected animals with a few drops of carbonated water. After that they were immediately picked onto Utermöhl microscopy chambers to a small drop of particle free seawater with a Pasteur pipette and the number of ingested microspheres was counted under an epifluorescence microscope."	The referenced study is based on various planktonic taxa found in the Baltic Sea. It is not clear from the study itself, or the context in which it is cited, whether these types of plankton exist in U.S. waters or whether there are similarities between the Baltic Sea planktonic taxa to and found in U.S. waters. In addition, the study uses fluorescent polystyrene particles. The study makes no attempt to determine if the planktonic taxa are attracted to the glow of those particles in the dark waters typically found in the oceans. In addition, the florescent polystyrene particles used are substantially smaller than particles derived from suspension polymerization PVC resins, which account for 95% of PVC resin produced in the U.S. Indeed, these particles range from 150-200 microns in diameter.
134	I.B.2.f, p. 17 FN 156 – Carson (2013)	. . to large, predatory sharks	The study suggests that fish of a variety of sizes attack drifting plastic with high frequency. The study states that marks made in 200 plastic items could be attributed to fish/sharks - i.e., the smallest marks could have been made by any number of small adult or juvenile fish in pelagic or coastal waters. The largest marks could correspond	The study examined the affinity of fish to attach and ingest drifting plastics; however, the specific gravity of PVC (1.35 to 1.45) does not allow the material to float in water, making it unlikely to be present on top of the water. The European Council of Vinyl Manufacturers, *Specific Gravity,* https://pvc.org/about-pvc/pvcs-physical-properties/specific-gravity/.

			o larger predatory sharks or fish (based on he width of the triangle teeth shapes)	
135	I.B.2.f, p. 17 FN 157 – Jacobsen (2010)	. . and endangered sperm whales (Physeter macrocephalus).	The study documents the mortality of two sperm whales associated with ingestion of arge amounts of marine debris which, to the best of their knowledge, constitutes a previously undocumented cause of anthropogenic mortality in this species. The majority of debris in each whale consisted of scraps of netting (81% dry weight), pieces of line (17%) and pieces of bags, made mostly of plastic (2%). The remaining debris included pieces of line of a variety of lengths and diameters, all of it made of floating polyethylene except for a few pieces of nylon line tied to floating net or line. There also was a variety of plastic garbage bag scraps and one large (0.9 kg) bag woven of narrow plastic strips.	The vast majority of derelict debris observed in the sperm whales' stomach contents during this study was netting. As indicated in the study referenced by CBD, plastic fish netting is made predominantly from nylon and polyethylene. The study identifies the remaining debris as floating polyethylene and a few pieces of nylon line. PVC debris was not identified.
136	I.B.2.f, p. 18 FN 158 – Auman (1997)	Nearly a decade ago, researchers found that over 97 percent of dead and injured Laysan Albatross (Phoebastria immutabilis) chicks contained plastics, and concluded that the incidence and quantity of ingestion was likely increasing.	Laysan Albatross chicks from Midway Atoll, North Pacific Ocean, were assessed in 1994 and 1995 for impacts of plastic ingestion. Masses and incidence of plastic in chicks were compared between birds found dead of "natural causes" and those injured by motor vehicles. Naturally killed Laysan Albatross chicks had significantly greater masses of plastic in their proventriculi and gizzards and had significantly lighter body masses and lower fat indices than injured but otherwise healthy chicks. Ingested plastic probably does not cause significant direct mortality in Laysan Albatross chicks, but likely causes physiological stress as a result of satiation and mechanical blockages. Of the 251 chicks studied, only six (2.4%) did not contain plastic. Plastic items comprised chips and shards of unidentified plastic, Styrofoam, beads, fishing line, buttons, chequers, disposable cigarette lighters, toys, PVC pipe and other PVC	The reference does not indicate how the plastics recovered from the chicks were identified. Since most of the plastic items were not identified by plastic type, it seems likely that the "PVC" items were only visually identified, not chemically tested for identify. In addition, there is no indication as to the amount or percentage of PVC that may have been ingested.

			ragments, golf tees, dish-washing gloves, nagic markers and caylume light sticks. The incidence and mass of plastic ingested by Laysan Albatross chicks appears to be ncreasing. In 1966 74% of 91 Laysan Albatrosses sampled at Pearl and Hermes Reefs (north-west Hawaiian Islands) contained, on average, 1.87 g of plastic, with eight pieces being the greatest number found in any individual (Kenyon and Kridler 1969). In 1983 on Sand Island, Midway Atoll, plastic contents in live and dead Laysan Albatross chicks averaged 35.7 g (39.3 cc) and 76.7 g (85.0 cc), respectively (Fry et al. 1987). An average of 46 cc of plastic was collected from Laysan Albatross chicks in 1986 and 5 cc in 1987, and the frequency of occurrence in both these years was 90% (Sileo et al. 1990). In the current study plastics were found in 97.6% of chicks sampled, and the masses recorded (23.8 g in dead chicks in 1994 and 18.1 g in 1995) are in the same order as those for the 1980s, which suggests an upward trend in concentrations of plastic debris on the surface of the north central Pacific Ocean since the mid-1960s.	
137	I.B.2.f, p. 18 FN 159 – Wright (2013)	Although most organisms seem to mistake plastic debris for natural prey or passively ingest particles during normal feeding behavior,	"A key factor contributing to the bioavailability of microplastics is their small size, making them available to lower trophic organisms. Many of these organisms exert limited selectivity between particles and capture anything of appropriate size (Moore, 2008). Alternatively, higher trophic planktivores could passively ingest micro-plastics during normal feeding behaviour or mistake particles for natural prey." This review focuses on marine invertebrates and their susceptibility to the physical impacts of microplastic uptake. Some of the main points discussed are (1) an evaluation	The referenced article mistakenly assumes that since PVC is produced at 19% of global plastic production, that it would be encountered by marine life in that same proportion to other plastics. This is an incorrect assumption since very little PVC s used in single use plastics of the type typically discarded and found accumulating in marine environments. In addition, the article indicates that fibrous microplastics appear to be the most abundant and even preferential for uptake by certain taxa. PVC is rarely used as a fibrous material, in contrast to marine netting which typically produced with nylon which has a specific gravity heavy enough for it to sink to the sediment levels.

			of the factors contributing to the bioavailability of micro-plastics including size and density; (2) an assessment of the relative susceptibility of different feeding guilds; (3) an overview of the factors most likely to influence the physical impacts of microplastics such as accumulation and translocation; and (4) the trophic transfer of microplastics.	
138	I.B.2.f, p. 18 FN 160 – Graham (2009)	certain species preferentially consume PVC and other plastic waste.	Here we show that four species of deposit-feeding and suspension-feeding sea cucumbers (Echinodermata, Holothuroidea) not only ingest small (0.25 mm<maximum dimension>15 mm) nylon and polyvinyl chloride (PVC) fragments along with sediment, but also ingest significantly more plastic fragments than predicted given the ratio of plastic to sand grains in the sediment. "Indeed, all species exhibited significantly greater ingestion of plastic fragments than expected for two of the three types of plastic we tested. This indicates that the sea cucumbers in our study selectively ingested plastic particles over sand grains and other particles present in the sediment. . . The voluntary ingestion of plastic particles by holothurians has significant ecological and ecotoxicological implications. First, although the innovative work of Thompson et al. (2004) demonstrated that an amphipod (Orchestia gammarellus), barnacle (Semibalanus balanoides), and polychaete (Arenicola marina) ingest plastic fragments in the lab, our results indicate that some holothurians selectively ingest plastics over sediment grains." PVC shavings, nylon line fragments, and PVC pellets were mixed with sand grains and sea cucumbers were found to selectively ingest plastic particles relative to the sand grains (the amount ingested was not	The data presented in this paper stem from a contrived study wherein only shaved PVC particles were mixed with sand and fed to the sea cucumbers in a laboratory setting. In another feeding, only cut nylon fibers were mixed with sand and fed to the sea cucumbers. In the PVC shaving experiment, the feedings where PVC was ingested by four different taxa ranged from 70 to 86% and the number of particles ingested ranged from 93 to 4,168 pieces (Table 3A). In the nylon line experiment, the feedings where nylon was ingested by four different taxa ranged from 86 to 99% and the number of particles ingested ranged from 232 to 5,147 pieces (Table 3B). If anything, the data shows a preference for nylon by the sea cucumbers because they eat it more often and eat more of it. Even though the study also took sediment samples from the three locations where the sea cucumbers were obtained, they did not provide any analysis of the composition of plastics found in the sediment.

			consistent with the ratio of plastic particles to sand grains in the sediment). The plastic particles had a larger surface area relative to the sand grains and the smooth surface prevented sand grains from adhering, allowing particles to be separated from the sediment. There is no indication that the composition of the plastic particles had an effect on the study. The study states that it is unknown if plastic ingestion adversely affects the physiology or fitness of holothurians. A dissecting microscope was used to determine the total number of sand grains defecated, and the size, shape, and quantity of plastic defecated. All sand grains were counted individually, unless the sea cucumber defecated over 1.0 g (dry weight) of sand, in which case the sand was dried, weighed, and 1/10th of the weight was extracted and counted. This quantity was multiplied by ten to approximate the total number of sand grains in the sample. In such cases, all of the defecated material was still searched carefully for plastic pieces. Ingested PVC pellets were simply counted. Nylon fishing line fragments were counted and measured to the nearest 0.25 mm. PVC shavings were counted, and dimensions measured to the nearest 0.25 mm.	
139	I.B.2.f, p. 18 FN 161 – Browne (2008)	Scientific evidence demonstrates that ingested plastics can remain in an organism's body for weeks, accumulating in the digestive tract or translocating to the circulatory system, thereby facilitating trophic transfer and increasing the risk that phthalates and other toxic chemicals will migrate into the organism's tissues.	Parenthetical: (noting that "particles of plastic have been shown to accumulate in the gut cavity of birds, fish, and polychaete worms," and reporting data indicating that plastic particles "translocated from gut cavity [of mussels] to the circulatory system in as little as 3 days and persisted in the circulatory system for over 48 days") *Mytilus edulis* ingested and transported particles of microplastic to the gut, where they accumulated in the digestive cavity and tubules (Hypothesis I). Though particles of	The study only assessed ingestion, translocation, and accumulation of microplastics in mussels. Accordingly, the study is not broad enough to support CBD's broad characterization of "organisms." In addition, no PVC particles were assessed in this study.

			plastic have been shown to accumulate in the gut cavity of birds, fish, and polychaete worms, translocation from the gut to the circulatory system of an invertebrate has not been previously shown. In our study particles of polystyrene translocated from the gut cavity to the circulatory system in as little as 3 days and persisted in the circulatory system for over 48 days (Hypothesis II). This is important because previous research investigating the ingestion of macroplastic debris (>1 mm) by marine animals has only showed that it may be retained in the digestive tract or egested in the form of feces. In the first experiment to determine whether mussels were able to uptake microplastic from the water column into their gut cavity, animals were exposed for 12 h to separate treatments (each with three replicates) containing 0.51 gL-1of either 2 μm fluorescently labeled microspheres of poly-styrene (excitation 365 nm/emission 447 nm; Brookhaven International, UK), nonlabeled (4-16 μm) microspheres of polystyrene (Sigma-Aldrich, UK), or a control treatment without plastic. After exposure, histological techniques were used to determine the presence of microplastic in the gut. A second experiment was conducted to establish whether ingested microplastic could translocate from the gut cavity to the circulatory system. Clean glass beakers (400 mL) were filled with 350 mL of filtered seawater, stirring bars were added, and the beakers were placed on magnetic stirrers. Mussels were randomly assigned to their time/treatment/replicate combination using a replicated block-design. Two sizes of fluorescent polystyrene particles, 3.0 μm (490 nm excitation/515 nm emission) and	

			9.6 µm (520 nm excitation/580 nm emission) were obtained from Molecular probes (USA) in a distilled water carrier. These particles are colored as part of manufacture through adsorption. In our exposure study we used 15,000 individual polystyrene microspheres for each plastic treatment (verified by Coulter Counter analysis) of either small or large size particles per mussel, in each time/treatment/replicate combination. The size of plastic chosen was representative of the smaller size range of particles known to be ingested by marine invertebrates (3, 5, 25) and the smallest diameter of plastic debris found in marine habitats (3). Mussels were exposed to these treatments for 3h, as preliminary work demonstrated that this was sufficient for microspheres to be ingested. Animals were then transferred to separate clean beakers and fed daily on I. galbana. Seawater was changed every other day. To check for the presence of polystyrene microspheres in fecal matter, seawater was vacuum filtered using Whatman GFA filters which were subsequently examined under fluorescence microscopy.	
140	I.B.2.f, p. 18 FN 162 – Farrell (2013)	Indeed, a recent study demonstrated that shore crabs (Carcinus maenas) ingest and retain plastic fragments originally consumed by prey.	This study investigated the trophic transfer of microplastic from mussels to crabs. Mussels (Mytilusedulis) were exposed to 0.5 mm fluorescent polystyrene microspheres, then fed to crabs (Carcinus maenas). Tissue samples were then taken at intervals up to 21 days. The number of microspheres in the haemolymph of the crabs was highest at 24 h (15 033 ml^{-1} ± SE 3146), and was almost gone after 21days (267 ml^{-1} ± SE 120). The maximum amount of microspheres in the haemolymph was 0.04% of the amount to which the mussels were exposed. Microspheres were also found in the stomach, hepato-pancreas, ovary and gills of the crabs, in decreasing	The referenced study was conducted with an extremely small variety of polystyrene beads (0.5 microns) and showed only a small retention of microspheres in the crabs that consumed mussels exposed to microplastics. Indeed, the study highlights the diminishing amount of microplastics up the food chain from trophic transfer with only 0.28% of the estimated ingested amount in the crab after 24 hours. Most of this material was egested within 21 days. Accordingly, CBD's statement is misleading and should be qualified with information about the diminishing retention rate and probability of transfer up the trophic chain.

			umbers over the trial period. This study is he first to show 'natural' trophic transfer of nicroplastic, and its translocation to aemolymph and tissues of a crab. Although the amount of microplastic that ransferred from mussels to crabs was small, his study has demonstrated that trophic ransfer occurs between mussels and crabs, nd that micro-plastic can translocate to the aemolymph and tissues of the crab. Comparison to another study seems to show hat the levels were much lower than xpected: "Kach and Ward (2008), showed hat mussels had a 14% retention efficiency for 0.5 mm fluorescent polystyrene nicrospheres. In this study, the mussels vere exposed to an estimated 411 million nicrospheres. At 14% retention efficiency, hey would retain 57.54 million nicrospheres. It was calculated that the umber of microspheres in the crab's entire aemolymph at 24 h was 163 111 ± 34 140 nicrospheres. This is 0.04% of the number of microspheres to which the mussels were xposed and 0.28% of the estimated number of microspheres retained by the mussels."	
141	I.B.2.f, p. 18 FN 162 – Murray (2011)		*Parenthetical*: (reporting that commercially-harvested Norway lobsters (*Nephrops orvegicus*) accumulated plastic transported by prey items) The aim of this study was to determine the xtent Nephrops consumes plastics in the Clyde Sea and if this intake occurs through heir diet. Plastic contamination was found o be high in Nephrops, 83% of the animals ampled contained plastics (predominately filaments) in their stomachs. Raman pectroscopy indicated that some of the nicrofilaments identified from gut contents ould be sourced to fishing waste. Nephrops fed fish seeded with strands of	The referenced study does not mention PVC but rather polyethylene, polypropylene, and nylon plastics, which are widely used in the sampling region for fishing nets.

			polypropylene rope were found to ingest but not to excrete the strands. The likely route for plastic found in Nephrops is via passive ingestion with sediment as they feed, or in the food itself (trophiclink).	
142	I.B.2.f, p. 18 FN 163 – Barakat (2014)	Moreover, new experimental evidence confirms what scientists have long suspected: additive chemicals transfer from plastics to organisms following ingestion.	*Parenthetical:* (finding that, after consuming PVC, rats exhibit symptoms similar to those associated with direct exposure to vinyl chloride) The PVC powder was dissolved in corn oil as a vehicle. Rats were divided into 3 groups each one contains 20 rats (Group I kept as positive control given corn oil only, group II given 1/20 LD50 and group III given 1/10 LD50). All rats dosed one dose/3 times /week (day after day), orally by using stomach tube for 2 months. At the end of first and second month, ten rats were sacrificed from each group and samples were collected. whole blood collected in clean dry centrifuge tubes, allowed to stand for one hour at room temperature till clotted and centrifuged at 3000rpm for fifteen minutes, for serum separation, then kept in (-20°C) for biochemical analysis. Tissue Specimens from liver were taken and immediately placed in clean sterile Eppendorf tube and preserved at (-80°C) for molecular study. This study revealed that the rats in both treated groups by PVC suffered from lethargy, anorexia, depression, rough hair coat, humped back posture and emaciation as shown in (Figures 1&2). These results similar to result obtained by Bi et al. [21] and Lee et al. [22]. These clinical signs may be attributed to toxic effect after administration of PVC and difference in the	This study was conducted in Egypt using PVC purchased from Sigma-Aldrich Company, Egypt. The PVC powder was dissolved in corn oil as a vehicle and delivered directly via a stomach tube. The authors do not indicate which type of PVC resin was used or the size of the particles. As such, the concentration of PVC resin ingested in unclear. Further, no mode of action was determined, as only serum levels were measured. As a result, it is unclear whether the increase in biomarkers results from ingestion of PVC or simply from ingestion of inert particles of that size.

			dose that high dose induced more rapid changes in the degree of toxicity.	
143	I.B.2.f, p. 18 FN 163 – Browne (2013)		*Parenthetical:* (presenting "the first suitable controlled experimental evidence showing that eating of plastics can move pollutants and additives into the tissues of animals") Here we show that pollutants and additives transfer via desorption from both sand and microplastics to the tissues of an important bioengineer. This is the first suitably controlled experimental evidence showing that eating of plastics can move pollutants and additives into the tissues of animals. We exposed lugworms (Arenicola marina) to sand with 5% microplastic that was presorbed with pollutants (nonylphenol and phenanthrene) and additive chemicals (Triclosan and PBDE-47). Microplastic transferred pollutants and additive chemicals into gut tissues of lugworms, causing some biological effects, although clean sand transferred larger concentrations of pollutants into their tissues. When the bioavailability of pollutants from sand and PVC was compared, larger concentrations transferred from the sand to lugworms. Thus, the extent and rate of desorption from sand was much greater than from plastic, which retained more of each pollutant than clean sand. It is, however, premature to conclude that, compared to plastic, sediment in habitats will transfer more pollutants into animals upon ingestion. Longer-term experiments are needed to compare retention in sediments containing more clay and organic carbon with that of smaller-sized polymers. For instance, polyethylene, polypropylene, and polystyrene debris in habitats have larger concentrations of organic pollutants than PVC [24], while smaller (e.g., <10 mm)	The data generated by this study does not support CBD's claim with respect to PVC. Indeed, the study indicates that larger concentrations of pollutants transferred from sand to lugworms than from presorbed PVC: "Despite particles of PVC containing 135% (nonylphenol) and 5,860% (phenanthrene) larger concentrations than those on sand (Figures 1A and 1B), worms exposed to sand (with smaller concentrations of pollutants) accumulated >250% more phenanthrene and nonylphenol in their tissues than when PVC transferred the pollutants." The study goes on to state: "[f]or instance, polyethylene, polypropylene, and polystyrene debris in habitats have larger concentrations of organic pollutants than PVC [24], …" suggesting that other types of plastics may be more prone to transfer adsorbed pollutants.

			nicroplastic translocate and accumulate in :ells and tissues of animals. Thus, certain plastics, at smaller sizes, could transfer :hemicals into the tissues directly, without :he need for gastric desorption. For now, our short-term experiments with arge proportions of PVC (5%) show that vorms eating microplastic accumulated arge enough concentrations of pollutants or additives to reduce survival (Triclosan), feeding (Triclosan and PBDE), immunity (nonylphenol), and antioxidant capacity (PVC). The PVC finding is based on the following: Because mammalian cells exposed to nanometer-sized plastic produce reactive oxygen species [35, 36] and lugworms use antioxidants in their tissues to buffer the oxidative damage caused by hydrogen peroxide that accumulates in tissues during summer low tides [37, 38], we measured the oxidative status of lugworms. Here we show that the coelomic fluid of lugworms that ngested sediment with PVC had >30% smaller capacity to deal with oxidative stress (Figure 3N), while exposure to pollutants and additives through desorption from PVC had no effect.	
144	I.B.2.f, p. 18 FN 163 – Cole (2013)		*Parenthetical:* (reporting that "[t]he leaching of additives and disassociation of toxic chemicals post-ingestion has been modeled in polychaete worms and demonstrated in streaked shearwaters") Here, we show that microplastics are ngested by, and may impact upon, zooplankton. We used bioimaging techniques to document ingestion, egestion, and adherence of microplastics in a range of zooplankton common to the northeast Atlantic, and employed feeding rate studies to determine the impact of plastic detritus on	This study investigated the ingestion of minute microplastics, ≤31 µm diameter, by a range of zooplankton species using polystyrene beads. The range of particle size polystyrene resin s well below that for PVC suspension resin type, which comprises over 95% of PVC resin produced. What this study shows is that larger size microplastics are not selectively ngested by the taxa studied. So, for an impact to be valid for this type taxa, very small particle sizes (7.3 micron) must be available which naturally excludes most PVC resin. The attrition of PVC into those small of particle size is not demonstrated nor explained in the study.

			ılgal ingestion rates in copepods. Using fluorescence and coherent anti-Stokes Raman scattering (CARS) microscopy we identified that thirteen zooplankton taxa had the capacity to ingest 1.7–30.6 µm polystyrene beads, with uptake varying by taxa, life-stage and bead-size. Post-ingestion, copepods egested faecal pellets laden with microplastics. We further observed microplastics adhered to the external carapace and appendages of exposed zooplankton. Exposure of the copepod Centropages typicus to natural assemblages of algae with and without microplastics showed that 7.3 µm microplastics (>4000 mL−1) significantly decreased algal feeding. Our findings imply that marine microplastic debris can negatively impact upon zooplankton function and health. Prolonged gut-retention times of plastics and gut-blockages in zooplankton may limit the ability of these organisms to ingest and digest food, and may pose a toxic risk. During manufacture, a suite of additives (e.g., plasticisers, flame-retardants, antimicrobials) are added to plastics, and large surface area to volume ratio and hydrophobic properties of microplastics make them particularly susceptible to the adherence of waterborne contaminants (e.g., PCBs, DDT, and PAHs). The leaching of additives and disassociation of toxic contaminants post-ingestion has been modeled in polychaete worms and demonstrated in streaked shear-waters.	
145	I.B.2.f, p. 18 FN 163 – Rochman (2013)		*Parenthetical:* (finding that fish absorb chemical constituents and other pollutants from ingested plastic debris) Fish were exposed to three treatments: a negative control (no LDPE), a virgin-plastic (LDPE virgin pre-production plastic) and a	This study actually found that there was a greater concentration of PBTs in fish exposed to plastic that was exposed to seawater for 3 months, than in fish exposed to virgin plastic. There was no statistically significant difference between the virgin-plastic and control treatments [i.e., 0% plastic] for certain contaminants. Accordingly, the study concludes that the plastic was absorbing contaminants from

			narine-plastic treatment (LDPE deployed in ın urban bay). Medaka were exposed to 10% plastic (by weight) mixed into reatment diets and sprinkled at the top of each tank. Diet and plastic dis- sociated at he surface and thus fish were exposed to plastic similar to the way they are in the wild (i.e. floating in the water column). As such, this translates to 8 ng of plastic per nL of water. Maximum concentrations eported in the North Pacific Subtropical Gyre are 300 ng/mL5, and thus the concentrations of plastic used in this experiment may be considered environmentally relevant. Our chemical analyses targeted polycyclic aromatic hydrocarbons (PAHs), PCBs and PBDEs see Figure 1 for a schematic diagram). 'Overall, we conclude that polyethylene ngestion is a vector for the bioaccumulation of PBTs in fish, and that toxicity resulting from plastic ingestion is a consequence of both the sorbed contaminants and plastic material. Thus, hazards related to plastic debris are not one-sided – supporting the dea that the mixture of plastic and sorbed pollutants associated with plastic debris should be acknowledged in aquatic habitats." 'Because there were not statistically significant differences among concentrations of PAHs, PCBs or PBDEs between the virgin-plastic and control reatments, we conclude that the PBTs sorbed to plastic from ambient seawater did ransfer from plastic to medaka upon ngestion. Thus, results from this experiment suggest that a chronic exposure of plastic debris in nature may be a significant route of exposure for PBTs in wildlife despite globally contaminated habitats."	he seawater and thus, not necessarily exposing the taxa to contaminants they are not already exposed to by virtue of their presence in the sea. The study also notes that LDPE was chosen because it "has a greater affinity for organic contaminants than other mass-produced polymers, comprises the largest component of plastic production globally (29%) and is one of the most common polymers recovered as aquatic debris. As such, these esults (particularly those regarding the plastic serving as a vector) may not reflect other plastics." To that end, LDPE also has a lower specific gravity (0.94) than PVC (1.35 to .45) meaning that the material will float and be more available to surface feeding fish than PVC would.

146	I.B.2.f, p. 18-9 FN 163 – Gaylor (2012)		*Parenthetical:* (concluding that soil-dwelling insects may "accumulate appreciable…burdens" of additive chemicals as a result of plastic ingestion) Our results indicate that soil-dwelling insects may ingest polyurethane foam present in current-use and derelict consumer products within human spaces and waste disposal sites and accumulate appreciable PBDE burdens. Crickets and other insects/arthropods may there- fore be underappreciated vectors of PBDE transfer from consumer products into terrestrial food webs. After usage, most PUF products end up in landfills and automotive dumpsites, etc. PUF products are also illegally discarded alongside roads and highways in some rural environments (Matthews County, VA Board of Supervisors Personal Communication). Soil insect abundance and activity is typically high in such waste disposal environments (Robinson, 2005) increasing opportunities for interaction with derelict consumer products and exposure to chemical additives therein.	Study focuses on the uptake of polybrominated diphenyl ether (PBDE) flame retardants via ingestion of polyurethane foam (PUF) products. Although the study may serve as an example of the accumulation of contaminants via plastic ingestion, it is irrelevant to PVC plastics because PBDE flame retardants are not included in PVC products.
147	I.B.2.f, p. 19 FN 164 – Rochman (2014)	These data bolster field observations indicating that fish,	This is an examination of the relationship between the bioaccumulation of hazardous chemicals in myctophid fish associated with plastic debris and plastic contamination in remote and previously unmonitored pelagic habitats in the South Atlantic Ocean. Using a published model, we defined three sampling zones where accumulated densities of plastic debris were predicted to differ. Contrary to model predictions, we found variable levels of plastic debris density across all stations within the sampling zones. Mesopelagic lanternfishes, sampled from each station and analyzed for bisphenol A (BPA), alkylphenols, alkylphenol ethoxylates, polychlorinated	The study sampled fish for BPA, alkylphenols, alkylphenol ethoxylates, PCBs, and PBDEs from locations in the South Atlantic Gyre (from Brazil to South Africa). None of these substances are used in PVC compounds. The study also did not identify the type(s) of plastics collected in the Gyre. Given the specific gravity of PVC, it seems unlikely that PVC would've been collected.

			biphenyls (PCBs) and polybrominated diphenyl ethers (PBDEs), exhibited variability in contaminant levels, but this variability was not related to plastic debris density for most of the targeted compounds with the exception of PBDEs. We found that myctophid sampled at stations with greater plastic densities did have significantly larger concentrations of BDE#s 183 –209 in their tissues suggesting that higher brominated congeners of PBDEs, added to plastics as flame-retardants, are indicative of plastic contamination in the marine environment. For fish tissue, individual fish were homogenized and subsampled for individual analyses of BPA, alkylphenolics (4-nonylphenol, 4n-octylphenol, 4-nonylphenol monoethoxylate and 4-nonylphenoldiethoxylate), PCBs (CB#s 1–209) and PBDEs (BDE#s 7, 8, 10–13, 15,17, 25, 28, 30, 32, 33, 35, 37, 47, 49, 51, 66, 71, 75, 77, 79, 85, 99, 100,105, 116, 119, 120, 126, 128, 138, 140, 153, 154, 155, 166, 181, 183,190, 196, 197, 203, 204, 206–209). Extraction and analysis procedureswere in accordance with AXYS Method MLA-084 for BPA, AXYS MethodMLA-080 Rev 2 for alkylphenols and alkylphenol ethoxylates, AXYSMethods MLA-013 and MLA-010 (EPA Method 1668) for PCBs and AXYS Method MLA-013 and MLA-033 (EPA Method 1614) for PBDEs. Sample extracts for BPA and alkylphenolics were analyzed using a Waters 2690 (Milford, MA, USA) high performance liquid chromato-graph coupled to a triple quadrupole mass spectrometer with a Waters (Milford, MA, USA) Xterra C18MS column.	

148	I.B.2.f, p. 19 FN 165 – Tanaka (2013)	. . . Seabirds	This study analyzed polybrominated diphenyl ethers (PBDEs) in abdominal adipose of oceanic seabirds (short-tailed shearwaters, Puffinus tenuirostris) collected in northern North Pacific Ocean. In 3 of 12 birds, they detected higher-brominated congeners (viz., BDE209 and BDE183), which are not present in the natural prey (pelagic fish) of the birds. The same compounds were present in plastic found in the stomachs of the 3 birds. These data suggested the transfer of plastic-derived chemicals from ingested plastics to the tissues of marine-based organisms. "Such sporadic detection is consistent with our observations. These studies did not investigate the plastics in the digestive tracts or correlate the detection of BDE209 with plastic ingestion. However, van Franeker et al. (2011) found frequent ingestion of plastics by fulmars. Our results suggest that their detection of BDE209 can be explained by the ingestion of plastics. Thus, the transfer of plastic-derived chemicals to biological tissues may occur in many species of birds over a wide range. More observations of the relationship between plastic ingestion and tissue PBDEs are needed."	This study examined levels of PBDEs in abdominal adipose of oceanic birds. As indicated above, PBDEs are not used in PVC materials.
149	I.B.2.f, p. 19 FN 166 – Fossi (2012)	. . . And endangered fin whales accumulate contaminants, including phthalate plasticizers, as a result of exposure to plastic debris.	Measurements of phthalate concentrations in surface neustonic/planktonic and water column samples and measurements of phthalate concentrations in stranded fin whale specimens were collected on the coasts of Italy. In view of the presence of microplastics in the Mediterranean environment, the detection of plastic additives in the blubber of fin whales and the long lifespan of the species, fin whales appear to be chronically exposed to persistent and emerging contaminants as a result of microplastic	As an initial matter, the microplastics and neustonic/planktonic samples in this study are from the Pelagos Sanctuary in Mediterranean Sea (between France and Italy). As such, this data is not representative of the U.S. environment and is also unlikely to represent the same types or compositions of plastics that are manufactured in the U.S. Beyond that, we question the data for two additional reasons. First, no detectable amounts of DEHP were measured for microplastics collected in the water column samples, where more PVC microplastics would be expected below the surface. In addition, the analysis of the levels of phthalates in zooplankton shows extraordinarily high standard deviations which is an indication of the uncertainly of the dataset

			ngestion. In this context, the preliminary observations presented in this paper suggest that phthalates can serve as a tracer for the intake of microplastics in micro-litter and in plankton by fin whales.	because of the high variability of the individual sample measurements. For DEHP, many of the data points were at the limit of detection or limit of quantification, with only a very small number of detections. This is an apparent anomaly with the broader detection of MEHP.
150	I.B.2.f, p. 19 FN 167 – Rochman (2014)	Thus, scientific evidence strongly indicates that the consumption of PVC and other plastic waste constitutes an important vector of chemical additives into the marine food web.	"Because many plastic additives are ubiquitous in the environment, scientists have struggled to establish that the bioaccumulation of these substances results from ingestion, rather than some other form of exposure. See, e.g., Rochman et al., supra note 164, at 632. Despite the surprising lack of data, experts have hypothesized that plastic consumption spreads chemical contamination throughout the food web." See e.g., *id.* (concluding that existing evidence "suggests that the ingestion of plastic debris may be an important mechanism for the bioaccumulation of hazardous chemicals in wildlife")	As noted above, The study sampled fish for BPA, alkylphenols, alkylphenol ethoxylates, PCBs, and PBDEs from locations in the South Atlantic Gyre (from Brazil to South Africa). None of these substances are used in PVC compounds. The study also did not identify the type(s) of plastics collected in the Gyre. Given the specific gravity of PVC, it seems unlikely that PVC would've been collected.
151	I.B.2.f, p. 19 FN 167 – Galgani (2013)		*Parenthetical*: ("Ingestion of microplastic material . . . presents a route by which chemicals could pass from plastics into the food chain.") An emerging area of concern is the accumulation of microplastic fragments in the water column and in sediments (Thompson et al., 2004). Pieces of common polymers (including polyester, nylon, polyethylene and polypropylene) of less than 20 mm have been recorded in the marine environment worldwide (Barnes et al., 2009). Plastics are biologically inert. They degrade to tiny particles that probably stay in the marine environment for long periods. Because of their size they are available to a wide range of organisms including deposit feeders, filter feeders and scavengers (Thompson et al., 2004). If ingested, plastics release chemicals (such as nonyl-phenols, polybrominated diphenyl ethers, phthalates or bisphenolA) but also	As discussed in line 4, there is no reference to PVC in this study.

			sorb hydrophobic pollutants (including PCBs and DDT). These may be transferred to organisms and there is concern about subsequent adverse effects (Mato et al., 2001; Teuten et al., 2009). Ingestion of microplastic material, therefore, presents a route by which chemicals could pass from plastics into the food chain. More research is needed to establish the full environmental relevance and potential impact of these microparticles, notably on distribution, transport, degradation/weathering processes and sorption/release mechanisms.	
152	I.B.2.f, p. 19 FN 167 – Davison (2011)		*Parenthetical*: ("[Ingestion of plastics by fishes may] serve as a point of entry of plastic-associated toxins into the food chain.") During the 2009 Scripps Environmental Accumulation of Plastics Expedition, we investigated whether mesopelagic fishes ingest plastic debris. A total of 141 fishes from 27 species were dissected to examine whether their stomach contents contained plastic particles. The incidence of plastic in fish stomachs was 9.2%. The ingestion rate of plastic debris by mesopelagic fishes in the North Pacific is estimated to be from 12,000 to 24,000 tons yr^{-1}. This study examines the effect of net feeding on the apparent rate of plastic ingestion by deploying multiple types of nets that capture different concentrations of plastic debris and by conducting an experiment to directly measure the incidence of net feeding.	PVC is not referenced in this study's exploration of plastic ingestion in the North Pacific.
153	I.B.2.f, p. 19 FN 167 – Barnes (2009)		*Parenthetical*: ("Small and microscopic plastic fragments present a likely route for the transfer of [additive] chemicals.") Documents global plastics production and the accumulation of plastic waste. "Small and microscopic plastic fragments present a likely route for the transfer of these chemicals because they have a much greater	As indicated above, CBD's reliance on this article is beyond general views on plastic waste. PVC is only mentioned twice in the context of construction and debris waste generation and the fraction of PVC in household waste, which are not relevant to CBD's comment.

			surface area to volume ratio than larger items of debris from which they have originated and because of their size they are available to a wide range of organisms, including deposit feeders such as the lug worm, Arenicola marina, that feed by stripping organic matter from particulates (Mayer et al. 1997; Voparilet al. 2004)."	
154	I.B.2.f, p. 19 FN 168 – Vethaak (2009)	After dissociating from PVC, phthalates accumulate in the tissues of aquatic organisms,	*Parenthetical*: (detecting nine phthalates and other endocrine-disrupting chemicals in the muscle tissue of wild-caught bream (Abramis brama) and European flounder (Platichthys flesus)) This study was carried out in the Netherlands on the occurrence of a number of estrogenic compounds in surface water, sediment, biota, wastewater, rainwater and on the associated effects in fish. Compounds investigated included natural and synthetic hormones, phthalates, alkylphenol(ethoxylate)s and bisphenol-A. The results showed that almost all selected (xeno-)estrogens were present at low concentrations in the aquatic environment. Locally, they were found at higher levels. Hormones and nonylphenol(ethoxylate)s were present in concentrations that are reportedly high enough to cause estrogenic effects in fish. Contaminant levels in both fish species did not correlate well with the external contaminant levels in the various environmental compartments of this study, which the author partly explains by the known rapid metabolism by fish of the compounds considered, and the likelihood that contaminant levels in fish represent exposure to xenoestrogens at many more locations where the fish might have roamed instead of only at the location where the fish are captured and the samples for chemical	As an initial matter, the samples that form the basis of this study were collected in the Netherlands in 1999. Accordingly, this information in quite dated and unlikely to be relevant to the U.S. Further, while many of the detected phthalates were used in non-PVC products such as cosmetics, lotions, soaps, air fresheners, paper products, adhesives, and coatings, the EU's REACH directive has since forced the phase-out of many of these phthalates. *See Phthalates*, ECHA, [HYPERLINK "https://echa.europa.eu/hot-topics/phthalates"]; *see also* ECHA, Annex XV Report: *An Assessment of Whether the Use of Ten Phthalates in Articles Should be Restricted in Accordance with Article 69(2) of REACH* (Apr. 4, 2022), https://echa.europa.eu/completed-activities-on-restriction (stating that "ECHA has not received any applications for authorisations for these [ten phthalates]. This indicates that the uses of these substances in articles have been largely phased out in the EU.)

			analysis of the abiotic compartments are taken.	
155	I.B.2.f, p. 19 FN 169 – Davison (2011)	. . . including those targeted by commercial fisheries.	*Parenthetical*: (observing that "[m]any commercially harvested fish feed at a high trophic level and may be subject to biomagnification of the toxins ingested by their prey")	As stated in line 153, this study does not analyze the type of plastic ingested, nor is it relevant to CBD claims about PVC.
156	I.B.2.f, p. 19 FN 169 – Chatterjee (2010)		*Parenthetical*: ("[Certain phthalates] can accumulate in the food chain via biomagnifications as one organism consumes food lower in the food chain and is subsequently consumed by an organism higher in the food chain; humans are generally at the top of such chains and this increases their exposure.") Butyl benzyl phthalate (BBP), an aryl alkyl ester of 1,2-benzene dicarboxylic acid, is extensively used in vinyl tiles and as a plasticizer in PVC in many commonly used products. BBP, which readily leaches from these products, is one of the most important environmental contaminants, and the increased awareness of its adverse effects on human health has led to a dramatic increase in research aimed at removing BBP from the environment via bioremediation. This review highlights recent progress in the degradation of BBP by pure and mixed bacterial cultures, fungi, and in sludge, sediment, and wastewater. Sonochemical degradation, a unique abiotic remediation technique, and photocatalytic degradation are also discussed. The degradation pathways for BBP are described, and future research directions are considered.	The bioaccumulation of phthalates is not the focus of this study, rather, it is the removal of BBP from the environment. BBP has not been widely used in PVC for decades, having been replaced by higher molecular weight phthalates.
157	I.B.2.f, p. 19 FN 170 – Latini (2004)	Scientific evidence indicates that humans acquire these chemicals primarily as a result of dietary exposure,	*Parenthetical*: explaining that dietary exposure is the main source of DEHP contamination among the general population General population may come in contact with DEHP by several sources of exposure,	Recent data from the National health and Nutrition Examination Survey shows that the metabolites for DEHP have diminished significantly during the period 2005 to 2020 because other higher MW plasticizers have been substituted for DEHP. These higher MW phthalates are correspondingly less volatile and less bioavailable to humans. *See* K. Chojnacka and M. Mikulewicz, *Bioaccumulation*

			but the main exposure is believed to be dietary, followed by indoor air.	Encyclopedia of Toxicology (3 Ed.) (2014), p. 456-460, https://www.sciencedirect.com/science/article/pii/B9780123864543010393.
158	I.B.2.f, p. 19 FN 170 – Trasande (2013)		*Parenthetical*: ("[D]ietary intake from contaminated food is the largest contributor of [DEHP] exposure in children.") Though medical devices (US Food and Drug Administration, 2012) and toys (Bouma and Schakel, 2002) can contain di-2-ethylhexylphthalate, dietary intake from contaminated food is the largest contributor to exposure in children (Schettler, 2006; US Agency for Toxic Substances & Disease Registry, 2012). Migration from di-2-ethylhexylphthalate-lined food packaging films appears to be the major route of contamination, though polyvinylchloride tubing (Petersen and Breindahl, 2000), gaskets in metallic caps for glass jars (Tsumura et al., 2002), and printing inks on labels (Cao, 2010) may also contribute.	This study is not related to discarded PVC products, but rather dietary exposure to DEHP. As a general matter, the U. S. Food and Drug Administration has studied the health effects of human consumption of phthalates and concluded there is not enough evidence to support any health restrictions for phthalates in contact with food items. *See* Katherine S. Carlos, Lowri S. de Jager & Timothy H. Begley (2018) Investigation of the primary plasticisers present in polyvinyl chloride (PVC) products currently authorised as food contact materials, Food Additives & Contaminants: Part A, 35:6, 1214-1222, DOI: 10.1080/19440049.2018.1447695 ("[h]owever, whether these observed health effects are a concern for humans has not yet been shown, and there are questions over how closely the reactions seen in mice and rats would mimic those in humans. There have been no studies to date which show any connection between human dietary exposure to phthalates and adverse health effects. Both the Centers for Disease Control and Prevention (CDC) and the National Institutes for Health believe that there is not enough data on the topic to decide whether low levels of phthalate exposure have any potential to cause problematic health effects in humans"). With respect to PVC items specifically, DEHP was replaced with DOA (Di 2-Ethylhexyl Adipate) in PVC food films around 2000.
159	I.B.2.f, p. 20 FN 171 – Cheng (2013)	. . . Including the consumption of contaminated fish and seafood.	"Consumption of freshwater fish and seafood is a major source of dietary intake of organic contaminants including phthalate esters (Chenet al., 2012) and OCPs (Dickman and Leung, 1998)."	Samples of fish were purchased from markets in Hong Kong, China for use in this study. Exposure via fish obtained in China is not representative of the U.S. environment, particularly as China's use of plasticizers differs significantly from that in the U.S.
160	I.B.2.f, p. 20 FN 172 - *Id.*	A recent analysis of marine and freshwater fish purchased from Hong Kong markets detected multiple phthalates in each sample, raising concerns that fish-heavy diets might lead to an increased incidence of cancer.	The bioaccessibility of phthalate esters in 20 fish species collected from Hong Kong market was evaluated using an in vitro gastrointestinal model. The ∑ phthalate ester concentration detected in fresh water fish ranged from 1.66 to 3.14 μg/g wet weight (ww) and in marine fish ranged from 1.57 to 7.10 μg/g ww, respectively. di-2-Ethylhexyl phthalate (DEHP) and di-n-butyl	Additionally, the study does not inform whether the fish were prepackaged at the fish market and somehow contaminated during handling, cleaning, and packaging. The fact that most fish contained measurable concentrations of each of the 12 phthalates analyzed for is remarkable, indicating a wide variability in potential for contamination from sources of phthalates including skin creams, air fresheners, lubricating oils, paper packaging, plastic packaging, and others. To that

			ɔhthalate (DBP) were the predominant compounds in both freshwater fish and narine fish. The digestible concentrations of ɔhthalate esters ranged from 0.20 to1.23 µg/g ww (mean 0.35 µg/g ww), and account for 2.44 to 45.5% (mean 16.8%) for raw concentrations of phthalate esters. In the present study, the accumulation ratio Rnn value of all phthalate esters was greater than 1 except for diisobutyl phthalate (DIBP), DBP and di-n-hexyl phthalate (DHP), suggesting that these phthalate esters could be accumulated during gastrointestinal digestion.	nd, it should be noted that laboratories are known to have ɔhthalate contaminants in the analytic equipment and therefore extreme care must be taken when analyzing for these substances, which are ubiquitous.
161	I.B.2.f, p. 20 FN 173 – Rozati (2002)	Similarly, in a study evaluating infertile men, researchers determined that phthalate concentrations were highest among regular fish-eaters, regardless of other lifestyle factors.	Purpose was to evaluate the role of the environmental estrogens polychlorinated biphenyls (PCBs) and phthalate esters (PEs) as potential environmental hazards in the deterioration of semen parameters in infertile men without an obvious etiology via a randomized controlled study. High performance liquid chromatography (HPLC) was used for extraction procedure and the Kolmogorov-Smirnov test was used for comparing semen quality among different categories of infertile men. PCBs were detected in the seminal plasma of infertile men but not in controls, and the concentration of PEs was significantly higher in infertile men compared with controls. PCB and PE concentrations were the highest in infertile urban fish eaters, followed by infertile rural fish eaters, infertile urban vegetarians, and infertile rural vegetarians. They were higher in infertile fish eaters than in infertile non–fish eaters regardless of the place of dwelling and were higher in infertile urban dwellers than in infertile rural dwellers regardless of the diet. 21 infertile men and 32 controls were used.	Study was conducted in India and is unrepresentative of the U.S. environment as India's use of plasticizers differs significantly from that in the U.S. In addition, the conclusion of the research suggests that the PCB's may be more responsible for the infertility than the PE's: "[f]rom the correlation studies it is evident that the PCBs have significant dose-dependent relationships with most of the semen parameters studied (ejaculate volume, total progressive motility, sperm vitality, osmoregulatory capacity, and sperm DNA integrity) when compared with Pes [phthalate esters], which suggests that the PCBs could perhaps be more instrumental than the PEs in the deterioration of semen parameters in infertile men without an obvious etiology."

162	I.B.2.f, p. 20 FN 174 – Abdel daiem (2012)	Scientific research reveals that phthalates also accumulate in terrestrial ecosystems,	*Parenthetical*: (observing that "[t]he use of sewage sludge in agriculture . . . poses a growing threat to ecosystems and human health," because it introduces phthalates into the food chain) This article describes the most recent methods developed to remove phthalic acid esters (PAEs) from water, wastewater, sludge, and soil. In general, PAEs are considered to be endocrine disrupting chemicals (EDCs), whose effects may not appear until long after exposure. There are numerous methods for removing PAEs from the environment, including physical, chemical and biological treatments, advanced oxidation processes and combinations of these techniques. This review largely focuses on the treatment of PAEs in aqueous solutions but also reports on their treatment in soil and sludge, as well as their effects on human health and the environment. It is common practice to dispose of biosolids by treating the sludge and using it for soil conditioning (biosolids represent half of European sludge production), and the resulting accumulation of persistent toxic organic compounds, as PAEs, in the soil poses a growing threat to ecosystems and human health. The use of sewage sludge in agriculture can produce human exposure during its application or through the resulting introduction of these compounds into the food chain. There is an urgent need to ensure that sludge is free from these contaminants before its utilization (Amir et al., 2005).	This article is not representative of U.S. landfills or wastewater treatment methods. All of the landfills identified and examined by the internal references in this article are in Europe. There are significant differences between landfill construction in Europe compared with those in the U.S. Furthermore, municipal wastewater treatment methods in the U.S. vary from those in Europe.
163	I.B.2.f, p. 20 FN 175 – Colacino (2010)	. . . contributing to the contamination of fruit, vegetables, meat, poultry, eggs and dairy products.	*Parenthetical*: (assessing the contribution of various foods to phthalate exposure) Study conducted an exploratory analysis of data collected as part of the 2003–2004	The relevance of the 2003–2004 National Health and Nutrition Examination Survey (NHANES) is diminished by its age. Indeed, current NHANES data for the U.S. population shows a significant continual reduction in phthalate metabolites since that time. BA Beckingham, *et al.*, *Phthalate*

			National Health and Nutrition Examination Survey (NHANES). Associations between dietary intake (assessed by a 24-hr dietary recall) for a range of food types (meat, poultry, fish, fruit, vegetable, and dairy) and phthalate metabolites measured in urine were analyzed using multiple linear regression modeling. Study found that metabolites of di- (2-ethylhexyl) phthalate (DEHP) and high-molecular-weight phthalate metabolites were associated with the consumption of poultry. Monoethyl phthalate, the metabolite of diethyl phthalate (DEP), was associated with vegetable consumption, specifically tomato and potato consumption. These findings from our study suggest that there is an association between dietary consumption of certain food types and levels of chemical contaminants measured in the NHANES study population. Poultry consumption was significantly associated with creatinine-adjusted DEHP metabolites MEHP, MEHHP, MEOHP, and MECPP as well as high-molecular-weight phthalate metabolites. Additionally, the finding that egg consumption is significantly associated with levels of MEHP suggests that chickens themselves may be contaminated with phthalates and that food is not being contaminated just through packaging and processing. Fruit and vegetable consumption was associated with metabolites of low-molecular-weight phthalates, such as DEP and DMP. Fruit consumption was inversely associated with metabolites of high-molecular-weight phthalates and DEHP metabolites.	*exposure among U.S. college-aged women: Biomonitoring in an undergraduate student cohort (2016-2017) and trends from the National Health and Examination Survey (NHANES, 2005-2016*), PLoS ONE 17(2): e0263578 (2022), https://doi.org/10.1371/journal.pone.0263578. In addition, the referenced study has significant caveats associated with its conclusions, for instance: "[d]ietary intake in this study was estimated by a single 24-hr dietary recall that estimated the food consumed the day before urine was collected, and chemical levels were measured on only one occasion. A follow-up study could benefit from multiple phthalate metabolite measurements to provide an average level. The possibility exists that these data may not be truly representative of either dietary intake or chemical levels…A large study, possibly government sponsored, of chemical contamination of food is likely necessary to accurately assess the amount of and sources of contamination to protect the health of the public."
164	I.B.2.f, p. 20 FN 175 – Fierens (2012)		*Parenthetical*: (explaining that phthalates present in raw cow's milk might derive from contaminated feed, including pasture plants)	This is an exposure-biomonitoring study and not potential exposure or effects from discarded PVC products.

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			As already mentioned in the previous sections, feed can be contaminated with DEHP as a result of migration from contact materials (Cao, 2010; CDC, 2009) or via environmental transfer (Blüthgen, 2003; Cousins and Mackay, 2003; Staples et al., 1997), which is exclusively the case for pasture. In this survey, the most relevant contamination pathways for eight phthalates (DMP, DEP, DiBP, DnBP, BBP, DEHP, DCHP and DnOP) were explored in raw cow's milk from different Belgian farms. Although DMP, DEP, DnBP, DCHP and DnOP were measured in various feed samples, they were not found in raw cow's milk, which might be due to the rapid metabolism of phthalates in cows. DEHP and to a smaller degree also DiBP and BBP concentrations in raw cow's milk varied across seasons and across farms, which reveals the influence of a seasonal variation in feed composition and the influence of using other feed products, disinfectants, cleaning agents, etc. at the farms. Additionally, phthalate containing contact materials that are being used during cultivation, transport, processing or during the milking process seem to be another important contamination pathway, since phthalate concentrations in manually obtained milk samples differed from concentrations in mechanically obtained milk samples. Furthermore, the results obtained in this survey confirm that the amount of DEHP in European cow's milk has decreased over the last decades.	As an initial matter, this is an exposure-biomonitoring study and does not address any potential exposure or effects from discarded PVC products. Specifically, this study explored the contamination of milk from 5 dairy farms in Belgium with 8 phthalates. The milking and storage process seems to have a significant impact on the results. Other potential contamination sources are from the use of a disinfectant during the inspection and cleaning of the cows' udders; cling films, sealants or tubes used for the production, mixing or storage at the farms; during the mechanical milking process as a result of migration from contact materials; migration from plasticized contact materials such as milk tubes or sealants; and from components in the cooling tanks. Notably, the data presented in Table 6 of the study show the steady decline in DEHP concentrations in cow's milk around Europe, which has declined by factors of 10 to 5 to 2 from 1990 to 2010.
165	I.B.2.f, p. 20 FN 175 – Ce-Hui Mo (2009)		*Parenthetical*: (explaining that vegetables accumulate phthalates "from soil-to-root transfer and subsequently root-to-shoot translocation.")	This study examined the concentrations of PAHs and PAEs in various vegetables collected from farms in South China. The study does not address potential exposure or effects from discarded PVC products or reflect the U.S. environment. Indeed, China's use of plasticizers differs significantly from that in the U.S.

			This study investigated the occurrence of 16 polycyclic aromatic hydrocarbons (PAHs) and 6 phthalic acid esters (PAEs) in 11 vegetable species collected from nine farms of the Pearl River Delta, South China. Twelve PAH compounds and all PAE compounds were detected by gas chromatography coupled with mass spectrometry (GC-MS) in vegetables. Samples were collected from and materials were purchased in China. Sample extraction and cleanup were performed according to USEPA methods 3550B and 3630C with modification, respectively. The accumulation of PAEs in vegetables mainly resulted from soil-to-root transfer and subsequently root-to-shoot translocation, while that of PAHs mainly derived from atmospheric deposition or foliar uptake from the air (Kipopoulou et al. 1999; Tao et al. 2006).	Even if the study were relevant to the U.S., it suggests that PAHs present a larger concern PAE's: "It should be noted that the BCFs (bioconcentration factor) for PAEs were generally lower than those for PAHs in the same sample, despite most samples having higher PAEs than PAHs. This might be attributed to their concentrations in farm soils, their physicochemical properties, and uptake and translocation mechanism by vegetables."
166	I.B.2.f, p. 20 FN 176 – Wormuth (2006)	In addressing the dangers associated with the bioaccumulation of PVC-derived chemicals, EPA must account for all sources of human exposure	*Parenthetical*: (analyzing various "oral, dermal, and inhalation pathways causing consumer exposure to phthalates") To find the important sources of phthalates in Europeans, a scenario-based approach is applied here. Scenarios representing realistic exposure situations are generated to calculate the age-specific range in daily consumer exposure to eight phthalates. The scenarios demonstrate that exposure of infant and adult consumers is caused by different sources in many cases. Infant consumers experience significantly higher daily exposure to phthalates in relation to their body weight than older consumers. The use of consumer products and different indoor sources dominate the exposure to dimethyl, diethyl, benzylbutyl, diisononyl, and diisodecyl phthalates, whereas food has a major influence on the exposure to	This is a review of multiple studies using various different methods; the studies may not be suitable for a meta-analysis. The authors attempted to assess phthalates by analyzing exposure to phthalates in daily life, not as a result of exposure to discarded PVC products. Based on European exposure and data is derived from EU risk assessments/studies.

			liisobutyl, dibutyl, anddi-2-ethylhexyl phthalates. The scenario-based approach hosen in the present study provides a link between the knowledge on emission sources of phthalates and the concentrations of phthalate metabolites found in human urine. To cover all relevant pathways, data from a variety of sources of different quality had to be used. For most input parameters, minimum, mean, and maximum values or 5th, median, and 95th percentile values are determined, depending on the quality of available data. For a few parameters only point estimates are used.	
167	I.B.2.f, p. 20 FN 177 – Colacino (2010)	. . . and consider the additive, synergistic and multiplicative toxic effects of other pervasive pollutants.	'Additionally, various countries have differing regulations about how much phthalate is allowed in a product, which can complicate exposure estimation. We recently described a mixture of chemicals, many with unknown to incompletely described human toxicity, found in U.S. foods (Schecter et al. 2009, 2010). The potential exists for additive, synergistic, or multiplicative toxic effects for chemicals found in food or food packaging, particularly for endocrine-disrupting compounds." Study conducted an exploratory analysis of data collected as part of the 2003–2004 National Health and Nutrition Examination Survey (NHANES). Associations between dietary intake (assessed by a 24-hr dietary recall) for a range of food types (meat, poultry, fish, fruit, vegetable, and dairy) and phthalate metabolites measured in urine were analyzed using multiple linear regression modeling. Study found that metabolites of di- (2-ethylhexyl) phthalate (DEHP) and high-molecular-weight phthalate metabolites were associated with the consumption of poultry. Monoethyl phthalate, the metabolite of diethyl phthalate	This is an exposure study based on an analysis of NHANES data and not potential exposure or effects from discarded PVC products. No part of this study examined additive, synergistic, or multiplicative toxic effects for chemicals found in food or packaging. Rather, the authors cite another work that indicates the potential for this should be studied. In addition, this study acknowledges an earlier comment by VI that laboratory contaminants of phthalates are a serious concern of any phthalate analysis, especially those not skilled in phthalate analysis: "Studying human exposure to phthalates is complicated by the difficulty in analyzing the parent compounds because of widespread environmental contamination, including contamination in laboratories, which makes determining how much of each phthalate is present in various products challenging." (P. 1002.)

		(DEP), was associated with vegetable consumption, specifically tomato and potato consumption.	
168	I.B.2.f, p. 20 FN 177 – Dorea (2008)		*Parenthetical*: (explaining that "human exposure to pollutants, when consuming fish or seafood, is rarely limited to a single chemical, especially when consuming large predatory species coming from a marine environment") Persistent substances such as MMHg and DHP are neurotoxic (Newland, 2002). Others such as PCB (Abdelouahab et al., 2008) and phthalate esters-PEs (Schoeters et al., 2008) cause thyroid and gonad hormone imbalance;). These substances accumulate in fish as a function of age (size), fat composition and length of food chain. Therefore, human exposure to pollutants, when consuming fish or seafood, is rarely limited to a single chemical (Muckle et al., 2001a, b), especially when consuming large predatory species coming from a marine environment (Grand-jean et al., 1995a).	This article discusses chemical, toxic, metabolic, and ecological characteristics associated with PBTs in fish. It does not specifically address phthalates or reference PVC.
§ I.B.2.g. Plausible Improper Management				
169	I.B.2.g, p. 21 FN 183 – NOAA Report on Marine Debris Sources, Impacts, Strategies & Recommendations (2008)	Experts attribute a large portion of marine plastic pollution to flawed waste management techniques,	The report considered persistent solid man-made debris from both land-based and ocean-based sources and its adverse impacts on the marine environment and navigation safety. "Debris can be the result of improper trash disposal, improper handling of materials, or inadequate reception facilities for waste. Litter, regardless of whether it is purposely or accidentally discarded or lost, has the potential to become marine debris. . . . Improperly disposed trash can wash into streams, combined sewer systems, and separate storm sewer systems (e.g., storm drains) and eventually be carried into coastal and ocean waters. . . . Overused and poorly managed landfill and transfer stations	The study discusses the impact of trash and debris in general and does not reference PVC specifically. It is important to underscore that PVC is not a major constituent of marine litter and is not widely used in single-use plastics type applications. As evidenced by EPA's Sustainable Materials Management (SMM) report, PVC comprises less than 3 % of plastic waste generated in the U.S. as municipal solid waste, which, as suggested by the referenced NOAA article, can serve as a source of marine litter. *See* U.S. EPA, Advancing Sustainable Materials Management: 2018 Tables and Figures (Dec. 2020), https://www.epa.gov/facts-and-figures-about-materials-waste-and-recycling/advancing-sustainable-materials-management.

			often can result in increased marine debris. Trash that is improperly covered during transport or deposition into landfills can be carried by wind into the marine environment or into other aquatic systems that transport the trash to the marine environment."	
170	I.B.2.g, p. 21 FN 184 – Barnes (2009)	. . . including the careless transport and improper burial of plastic trash.	The article focuses on the global plastics production and the accumulation of plastic waste. "There are many sources for plastics accumulating in the environment from direct dropping and dumping of litter on land or at sea to blowing from landfill sites, losses in transport and accidents." "Most waste plastics, including the large proportion used in single-use applications such as packaging, are disposed of in landfill sites. However, plastic persists in landfill sites and if not properly buried may later surface to become 'debris.'"	As stated previously, CBD's reliance on this article is for general views on plastic waste. There are only two limited mentions of PVC in the reference, which we identified in line 2 above. This includes Table 1 of the reference, which lists PVC at 6% of all plastic discarded as municipal solid waste (MSW) in the U.S. in 2005. Since that time, EPA's SMM Report (see line 169) has tracked the steady decline of PVC in MSW discards to the most recent level reported by EPA in 2020 at approximately 3% of all plastics.
171	I.B.2.g, p. 21 FN 185 – Chatterjee (2010)	Similarly, scientific evidence indicates that phthalate plasticizers may percolate into groundwater through poorly lined landfills or enter the atmosphere as a consequence of PVC incineration, resulting in widespread contamination.	"The review highlights progress in the degradation of BBP, which is reportedly used extensively in vinyl tiles and as a plasticizer in PVC, by pure and mixed bacterial cultures, fungi, and in sludge, sediment, and wastewater." "As plasticizers, phthalates are not tightly bound to the plastics, and they therefore leach from plastic products into the environment over time." "According to the American Chemistry Council (ACC, formerly CMA) (CMA 1999), the largest use of BBP is in vinyl tile. BBP is also used as a plasticizer in PVC-based food conveyor belts, carpet tiles, artificial leather, tarps, automotive trim, weather stripping, traffic cones, and to a limited extent in vinyl gloves (IPCS 1999) . . . As an environmental contaminant, BBP is usually released into the air in the form of	Poorly lined landfills present a generic problem and not one specific to PVC products. Even if that were the case, BBP is no longer widely used in PVC. This low molecular weight phthalate has been substituted with other higher molecular weight varieties that are far less likely to leach and are significantly less water soluble. As for combustion, the internally referenced studies that claim to detect phthalates in combustion byproducts are from the mid-1980's. U.S. EPA air emission regulations have strengthened significantly since the mid-1980's to a point that the studies referenced are no longer relevant.

			dust. BBP may be released to the air through the combustion of refuse (Graedel et al. 1986), and it has also been detected in stack emissions from hazardous waste combustion facilities and from coal burning power plants in the USA (Oppelt 1987). Once in the atmosphere, BBP spreads and can subsequently be detected in the atmosphere, soil, surface water, and sediments (CICAD 1999)."	
172	I.B.2.g, p. 21 FN 185 – Kang (2010)		The study looked at Diethyl phthalate (DEP)'s biochemical toxicity in aquatic animals. "DEP is known to be released into the environment during its synthesis processes, final utilization of DEP-containing products, or disposal of used products (Giam et al., 1978; Joblings et al., 1995). The most important release source to the aquatic environment is ascribed to leaching from landfill sites (Silva et al., 2004). Consequently, DEP has been widely identified in aquatic environments and their biota (Giam et al., 1978; Fatoki and Vernon, 1990)."	This is a toxicology study regarding potential effects of exposure to a substance and not potential exposure or effects from discarded PVC products. This referenced study was done in Korea and is not relevant to the U.S. No association is established between DEP leached from landfills and PVC products. Indeed, DEP is not an important or widely phthalate for PVC. It is mostly used in personal care products.
173	I.B.2.g, p. 21 FN 186 – Swan (2010) (Masculine Play)	Indeed, "virtually universal" human exposure may already have contributed to a variety of public health crises,	"Moreover, phthalates are present in so many other products and manufactured in such quantity, that exposure is virtually universal (CDC, 2005)."	As stated in line 61, the article does not establish a connection between discarded PVC products and phthalate exposure.
174	I.B.2.g, p. 21 FN 186 – Latini (2006)		The study explores evidence linking phthalate exposure and the decline of human male fertility, especially in developed countries and clarifies the exact role of PPARs activation in their adverse effect on the male reproductive tract. "Phthalates have been used as additives in industrial products since the 1930s, and are universally considered to be ubiquitous environmental contaminants. The general population is exposed to phthalates through	Study of possible link to endocrine disruption. The authors conducted a review in 2006 of evidence linking phthalates to a decline in male fertility. No reference to discarded PVC products or PVC.

			consumer products, as well as diet and medical treatments . . . Unprecedented declines in fertility rates and semen quality of antenatal origin have been reported during the last half of the 20th century in developed countries and increasing interest exists on the potential relationship between exposure to environmental contaminants, including phthalates, and human male reproductive health.”	
175	I.B.2.g, p. 21 FN 187 – Teitelbaum (2012)	including the increased incidence of obesity	The study examined associations between urinary phthalate metabolite concentrations and body size measures in children. Urinary concentrations of nine phthalate metabolites: monoethyl (MEP); mono-n-butyl(MBP); mono-(3-carboxypropyl) (MCPP); monobenzyl (MBzP); mono-isobutyl (MiBP); mono-(2-ethyl-hexyl) (MEHP); mono-(2-ethyl-5-oxohexyl) (MEOHP); mono-(2-ethyl-5-carboxypentyl) (MECPP); andmono-(2-ethyl-5-hydroxyhexyl) phthalate (MEHHP) and the molar sum of the low molecular-weight phthalate metabolites (low MWP: MEP, MBP and MiBP) and high molecular-weight phthalate metabolites (high MWP: MECPP, MEHHP, MEOHP, MEHP and MBzP) and of four di-(2-ethylhexyl) phthalate (DEHP) metabolites (SDEHP: MEHP, MEHHP, MEOHP, MECPP) and anthropometry, including body mass index and waist circumference were measured among 387 Hispanic and Black, New York City children who were between six and eight years at cohort enrollment (2004–2007). Relationships between baseline metabolite concentrations and body size characteristics obtained one year later were examined using multivariate-adjusted geometric means for each body size characteristic by continuous and categories of phthalate metabolite concentrations. Stratified analyses by body size (age/sex	As stated in line 75, this study concluded that the findings were not sufficient to attribute a causal effect for phthalates on increased body size. Beyond that, the association observed between phthalates and body size measurements were attributed to low molecular weight phthalates, which are not widely, if at all, used in PVC.

			specific) were conducted. 'Our main findings were associations of MEP and low MWP with BMI and waist circumference among overweight children. For example, there was a 2 unit increase in BMI and a 5cm increase in waist circumference for both MEP and low MWP going from the from 1st (<1µm) to 3rd (>3µm) micromolar biomarker quantiles. This corresponds approximately to a tenfold increase in concentrations (median MEP 100µg/gC in <1µm quantile and 1211µg/gC in >3µm quantile)."	
176	I.B.2.g, p. 21 FN 187 – Stahlhut (2007)		The study investigated phthalate exposure and its associations with abdominal obesity and insulin resistance. 'In the present study, we found that the log-transformed concentrations of several phthalate metabolites were positively and significantly correlated with abdominal obesity (MBzP, MEHHP, MEOHP, MEP) and insulin resistance (MBP, MBzP, MEP) in adult U.S. males." 'Subjects were adult U.S. male participants in the National Health and Nutrition Examination Survey (NHANES) 1999–2002. We modeled six phthalate metabolites with prevalent exposure and known or suspected antiandrogenic activity as predictors of waist circumference and log-transformed homeostatic model assessment (HOMA; a measure of insulin resistance) using multiple linear regression, adjusted for age, race/ethnicity, fat and total calorie consumption, physical activity level, serum cotinine, and urine creatinine (model 1); and adjusted for model 1 covariates plus measures of renal and hepatic function (model 2). Metabolites were mono-butyl phthalates (MBP), mono-ethyl phthalate (MEP), mono-(2-ethyl)-hexyl phthalate	This is a 2007 exposure-biomonitoring study of phthalates and not potential exposure or effects from discarded PVC products. The study is not specific to PVC or linked to plausible management of discarded PVC products.

			(MEHP), mono-benzylphthalate (MBzP), mono-(2-ethyl-5-hydroxyhexyl) phthalate (MEHHP), and mono-(2-ethyl-5-oxohexyl) phthalate (MEOHP)." "In this national cross-section of U.S. men, concentrations of several prevalent phthalate metabolites showed statistically significant correlations with abdominal obesity and insulin resistance. If confirmed by longitudinal studies, our findings would suggest that exposure these phthalates may contribute to the population burden of obesity, insulin resistance, and related clinical disorders."	
§ I.B.2.h. Quantities of Waste Generated				
177	I.B.2.h, p. 21 FN 189 – Am. Chemistry Council (2014)	Despite slow economic growth, United States manufacturers produced over 100 billion pounds of plastics in 2013, including nearly 15.5 billion pounds of PVC	A summary of U.S. resin production, sales & captive use 2013 vs 2012 from ACC. 107,522 million pounds plastic produced in 2013; 15,373 million pounds of PVC produced in 2013.	Sales and captive use data include imports; Canadian and Mexican production and sales data also included. This 2013 information is dated. More recent data shows that overall plastics produced in 2019 in the U.S. and Canada amounted to 121,457 million pounds. Of that, 15,931 million pounds of PVC was produced and 10,521 million pounds was consumed domestically. Amer. Chemistry Council, 2020 Resin Report.
178	I.B.2.h, p. 21 FN 190 – Wams (1987)	. . . and approximately 4.7 billion pounds of associated DEHP.	*Parenthetical*: (explaining that, on average, DEHP constitutes 30 percent of finished PVC) "The global production of DEHP amounts to 3-4 million tonnes (103kg) per annum. Figures for the U.S.A. and Europe indicate that each continent produces approximately one-third of the total production. . . Some 95% of all DEHP is used as a plasticizer in plastics; polyvinylchloride (PVC) utilizes 85% of the total production of plasticizers with DEHP being the most important plasticizer for PVC. It is also used as a plasticizer in cellulose ester plastics and synthetic elastomers. Plasticized PVC is used for car seats, furniture, bloodbags and tubes, vinyl floor and wall covering, cables and in foils for a wide variety of purposes, from packaging to building and	This article is from 1987. Based on 2019 data, the VI estimates that plasticizers use in general (not just DEHP) would amount to approximately 1,130 million pounds. (Refer back to calculations at line 85).

			construction. The DEHP content of these products varies depending on the application of the plastic, but, in general, it is between 20 and 40% of the weight, with 30% as a reasonable average."	
179	I.B.2.h, p. 21 FN 191 – Am. Chemistry Council (2014)	Experts expect these totals to increase in coming years, as "the surge in unconventional oil and gas development" lowers production costs, and the continued recovery of the construction industry and other important markets gives rise to increasing demand.	"The U.S. manufacturing sector, which represents the primary customer base for resins, is pulling out of a soft patch. Manufacturing growth slowed in 2013 largely due to the federal government sequester and to weakness in major export markets. However, the surge in unconventional oil and gas development is creating both demand side (e.g., pipe mills, oilfield machinery) and supply-side (e.g., chemicals, fertilizers, direct iron reduction) opportunities. Indeed, the enhanced competitive position with regard to feedstock costs will support U.S. chemical industry production going forward, with particular strength in plastic resins."	Domestic and world economies will continue to fluctuate. This passage referred to the impact of domestic shale oil production, which has shifted, and is not specific to PVC or discarded PVC products.
180	I.B.2.h, p. 22 FN 192 – EPA (2012)	According to EPA, PVC is "not widely recycled in practice."	"Life-cycle inventory data for other recycled plastic resins is not yet available, and some plastics (e.g., PVC) are not widely recycled in practice (EPA, 2011)."	In contrast to CBD's assertion, there are over 100 PVC recyclers in the U.S. that recycle approximately 1.1 billion pounds of PVC annually with about 13% being post-consumer. VI maintains a list of PVC recyclers on its website at: [HYPERLINK "https://www.vinylinfo.org/recycling-directory/"] . The VI estimates the PVC recycling rate in the U.S. to be 32.5%. *See* Krock, R., "Update on Vinyl Industry in U.S. and Canada", Proceedings of the IOM3 PVC2021 Conference (May 12, 2021). A more detailed discussion is provided in the main comments on PVC recycling.
181	I.B.2.h, p. 22 FN 193 – Belliveau (2004)	Indeed, estimates indicate that more than 7.2 billion pounds of this material enter landfills throughout the United States each year.	*Parenthetical*: (calculating, on the basis of existing data, that annual PVC disposal ranges up to 7.2 billion pounds, but observing that "[t]he amount of PVC [generated by the construction industry] may be seriously underestimated.") The document summarizes the total amount of PVC discarded annually in the US as municipal solid waste, medical waste, and	The VI's estimates for landfilled PVC products are much lower than CBD asserts. Using EPA's 2018 Sustainable Materials Management Report, some 1.4 billion pounds of PVC are landfilled as MSW. VI estimates that another 300 million pounds of discarded PVC are treated in waste to energy recovery facilities since no presorting is performed to remove PVC from those streams. VI also estimates some 1 billion pounds of PVC materials are disposed of in construction and demolition landfills annually.

			construction & demolition debris. The data, which appears to be based on data from 1993-2004, calculates the annual amount of PVC discarded annually in municipal solid waste, medical waste, and construction and demolition debris to be between 3.7 and 7.2 billion pounds.	
§ I.B.2.i. Nature and Severity of the Human Health and Environmental Damage that Has Occurred				
182	I.B.2.i, p. 22 FN 194 – Mulder (2001)	By the mid-1970s, vinyl chloride had contributed to nearly one dozen worker deaths,	"[In 1974,] B.F. Goodrich made public the fact that three employees had died from angiosarcoma during the past two years. In short order, another eight employee deaths were also ascribed to VC or vinyliden exposure [28–30]."	Mundt KA, Dell LD, Crawford L, et al., "Quantitative estimated exposure to vinyl chloride and risk of angiosarcoma of the liver and hepatocellular cancer in the US industry-wide vinyl chloride cohort: mortality update through 2013", Occup Environ Med 2017;74:709–716 provides the most recent study of ASL cases in the U.S. According to this study: "No clear exposure–response relationships were observed for mortality from other cancers, including lung cancer, brain cancer, non-Hodgkin's lymphoma or melanoma. And Risks of ASL and HCC were only elevated among workers with very high estimated cumulative exposures, that is, over 1000 parts per million-years, and after long latencies (median latency was 36 and 48 years, respectively, for ASL and HCC)." The total cohort of worker history studied were 9,951 men employed in the vinyl chloride industry covering health records from 1942 to 2013. Of this cohort, 63 cases of ASL, 32 cases of HCC and 36 unspecified cases of liver cancer mortality were confirmed."
183	I.B.2.i, p. 22 FN 195 – Kielhorn (2000)	. . . and experts warn that the continued widespread use of this substance "remains a cause for concern."	This article discusses how, from its view, VC remains a cause for concern because of continued exposure and reports of angiosarcoma of the liver. "As a result of the strict occupational exposure limits, no cases of angiosarcoma of the liver (ASL), the tumor associated with VC exposure, have been reported in new workers exposed to VC in those factories since that time. . . But recent epidemiologic, environmental, and biomechanistic findings have opened up new aspects of this chemical. . . VC	This review is based on studies from several countries with varying histories of worker safety protections and, even then, concludes that "Risk assessments derived from animal studies seem to overestimate the actual risk of cancer when comparing estimated and reported cases of ASL." Please refer to line 182 above for the most recent U.S. study of ASL cases (Mundt). According to the Mundt study, cases of ASL were limited to workers exposed to high levels of occupational exposure to vinyl chloride up until the regulations were enacted, and that other cancers were insignificant in the study group.

			remains a cause for concern because potential exposure to this chemical and new cases of ASL are still being reported." "Risk assessments derived from animal studies seem to overestimate the actual risk of cancer when comparing estimated and reported cases of ASL."	
184	I.B.2.i, p. 22 FN 197 – Latini (2006)	prenatal exposure to phthalate plasticizers and their metabolites interferes with hormone regulation and alters sexual development in male laboratory animals, inducing a suite of abnormalities known to scientists as "phthalate syndrome."	This article reviews the data that support or discounts the evidence existing to date linking phthalate exposure and the decline of human male fertility, especially in developed countries. "Many adverse effects on animal fertility and reproduction have been documented for phthalates following exposure before puberty. In particular, certain phthalate esters (DEHP, DBP, BBP) when administered to pregnant experimental animals during a critical window of development appear to play a relevant role in determining reproductive and developmental toxicity. These esters have been shown to produce a syndrome of reproductive abnormalities. The "phthalate syndrome" is characterized by malformations of the epididymis, vas deferens, seminal vesicles, prostate, external genitalia (hypospadias), cryptorchidism and testicular injury together with permanent changes (feminization) in the retention of nipples/areolae (sexually dimorphic structures in rodents) and demasculinization of the growth of the perineum resulting in a reduced anogenital distance (AGD), i.e. the distance from the anus to the base of the scrotum in males and from the anus to the base of the genitals in females."	This is a review of studies regarding potential reproductive health effects of exposure to phthalates and not potential exposure or effects from discarded PVC products. The Latini study singularly focuses on phthalates without considering other endocrine disrupting substances that males are widely exposed to which could contribute to declining sperm quality. To that end, the three predominant phthalates cited for impacting the development of the male reproductive tract (DBP, BBP, and DEHP) are less commonly used phthalates in the PVC industry. DBP and BBP are more commonly used iin personal care products.
185	I.B.2.i, p. 22 FN 198 – Frederiksen (2007)	Among humans, chronic contamination begins even before birth,	This article discusses the metabolism of phthalates in humans. "Little is known about these pathways, but recent studies have confirmed that unborn	This is a review of studies regarding potential prenatal exposure to phthalates and not potential exposure or effects from discarded PVC products.

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			ınd infants are in fact highly exposed to phthalates. In a model system, it was shown that monoester phthalates get distributed in cord, placenta, maternal and foetal perfusate in accordance with the physical-chemical properties of the compounds. An American study of samples from 54 anonymous donors has shown very low excretion of phthalate metabolites into amniotic fluids. In fact, out of ten measured phthalate metabolites only MEP, MPB and MEHP were found in amniotic fluid and the maximum levels of these three compounds were 9.0, 264 and 2.8 ng/mL, respectively [47]."	
186	I.B.2.i, p. 22 FN 199 – Swan (2005)	. . . posing grave biological consequences.	*Parenthetical*: ("[H]umans may be more sensitive to prenatal phthalate exposure than rodents.") The study presents data to examine AGD and other genital measurements in relation to prenatal phthalate exposure in humans. "The associations between male genital development and phthalate exposure seen here are consistent with the phthalate-related syndrome of incomplete virilization that has been reported in prenatally exposed rodents. The median concentrations of phthalate metabolites that are associated with short AGI and incomplete testicular descent are below those found in one-quarter of the female population of the United States, based on a nationwide sample. These data support the hypothesis that prenatal phthalate exposure at environmental levels can adversely affect male reproductive development in humans." "General linear models" were used to explore the relationship between phthalate metabolite concentration and genital parameters.	This is a review of studies regarding potential reproductive health effects of exposure to phthalates and not potential exposure or effects from discarded PVC products. As indicated above, the cited study is "the first study to look at subtle patterns of genital morphology in humans in relation to any prenatal exposure."

187	I.B.2.i, p. 22 FN 200 – Swan (2000)	Moreover, a growing body of experimental and epidemiological evidence suggests that widespread exposure to phthalates and other endocrine-disrupting chemicals has already contributed to significant declines in semen quality	This discusses prior analysis of studies on sperm density in relation to newer, current studies (uses 54 of the 61 studies previously analyzed, and added 47 English language studied published between 1934-1996). 'In 1992 Carlsen et al. stated that . . . reports published worldwide indicate clearly that sperm density has declined appreciably during 1938-1990.' Subsequently, this conclusion has been supported by findings from some studies, but not by others. . . The current analysis suggests that the previously reported trends have continued, at least until 1996." (internal citations omitted) It seems that, due to the variability between studies, they had to determine how to find/calculate similar trends across all of them.	No direct link to PVC. These references are reviews of studies regarding potential reproductive health effects of exposure to a substance and not potential exposure or effects from discarded PVC products. More recent studies suggest that sperm count varies within a wide range, much of which can be considered non-pathological and species-typical. This hypothesis, known as the sperm count biovariability (SCB) hypothesis, should be weighed against the prior studies cited. *See* Boulicault, M. et al., "The future of sperm: a biovariability framework for understanding global sperm count trends", Human Fertility, May 10, 2021.
188	I.B.2.i, p. 22 FN 200 – Fisher (2004)		*Parenthetical*: (reporting that 48 percent of young Danish men reporting for military service between 1996 and 1998 exhibited sperm counts associated with impaired fertility, while one-quarter qualified as 'abnormal," according to World Health Organization guidelines) This review aims to give a brief overview of the issues surrounding the perceived decline in human male reproductive health and the importance of the hormonal environment for the development of the testis and reproductive tract. The consequences for the male reproductive tract of abnormal androgen levels or action are discussed with reference to environmental anti-androgenic compounds In contrast to the cited purpose, the review states that "[t]here is currently no strong data to suggest that environmental EDCs are responsible for the observed degeneration in	

			human male reproductive health, but there are secular trends to suggest that it is declining."	
189	I.B.2.i, p. 22 FN 201 – Latini (2006)	. . . and deteriorating reproductive health across the industrialized world	This article reviews the data that support or discounts the evidence existing to date linking phthalate exposure and the decline of human male fertility, especially in developed countries. "Unprecedented declines in fertility rates and semen quality of antenatal origin have been reported during the last half of the 20th century in developed countries and increasing interest exists on the potential relationship between exposure to environmental contaminants, including phthalates, and human male reproductive health."	As mentioned above at line 184, this study singularly focuses on phthalates without considering other endocrine disrupting substances that males are widely exposed to which could contribute to declining sperm quality. To that end, the three predominant phthalates cited for impacting the development of the male reproductive tract (DBP, BBP, and DEHP) are less commonly used phthalates in the PVC industry. DBP and BBP are more commonly used in personal care products.
190	I.B.2.i, p. 22 FN 202 – Fisher (2004)	Within the past decade, researchers have identified a spectrum of increasingly prevalent disorders, collectively termed "testicular dysgenesis syndrome," which likely arise from impaired hormone production during fetal development.	This review aims to give a brief overview of the issues surrounding the perceived decline in human male reproductive health and the importance of the hormonal environment for the development of the testis and reproductive tract. The consequences for the male reproductive tract of abnormal androgen levels or action are discussed with reference to environmental anti-androgenic compounds "This has led to the proposal that low sperm counts, hypospadias, cryptorchidism and testicular germ cell cancer are interrelated disorders comprising a 'testicular dysgenesis syndrome' (TDS; see Fig. 1) (Skakkebaek et al. 2001, Sharpe 2003). The disorders that comprise TDS all have their roots in fetal development, suggesting that a possible causal link lies in abnormal hormone synthesis or action during reproductive tract development. From the historical literature, it is well known that the administration of diethylstilboestrol (DES; a potent synthetic oestrogen) to pregnant humans and rodents causes reproductive	As discussed in line 187, more recent studies suggest that sperm count varies within a wide range, much of which can be considered non-pathological and species-typical. This hypothesis, known as the sperm count biovariability (SCB) hypothesis, should be weighed against the prior studies cited. *See* Boulicault, M. et al., "The future of sperm: a biovariability framework for understanding global sperm count trends", Human Fertility, May 10, 2021. No connection between impaired hormone production during fetal development to discarded PVC products.

			ract abnormalities in the offspring (Stillman 1982)."	
191	I.B.2.i, p. 23 FN 203 – Howdeshell (2008)	Specific symptoms, including genital malformations, such as undescended testicles and displaced urethras, poor semen quality and testicular cancer, mirror the effects of prenatal phthalate contamination in laboratory animals,	The study reviews evidence that rat reproductive development is affected by phthalate esters, summarizes research on the cumulative effects of binary and complex mixtures of phthalates with each other and/or with other androgen-disruptive chemicals on male reproductive development, and discusses how laboratory animal research regarding the effects of phthalate esters on reproductive development furthers our understanding of the potential health risks of such chemicals on the developing human. "Male laboratory rats exposed in utero to certain phthalate esters display malformations and alterations of reproductive tissues indicative of a suppression of fetal testicular testosterone and insulin-like 3 hormone (insl3) production (Foster, 2006; Gray et al., 2000; Wilson et al., 2004) and multinucleated germ cells (Ferrara et al., 2006; Foster, 2006; Parks et al., 2000; Scott et al., 2007). Environmental chemicals, such as the phthalate esters, have been suggested as potential causal agents of human testicular dysgenesis syndrome (TDS), which bears striking similarity to the reproductive malformations, decreased sperm abundance, and histopathological changes in rat testes following in utero exposure to certain phthalate esters (Skakkebaek et al., 2001; Virtanen et al., 2005)."	This is a review of studies regarding potential reproductive health effects of exposure to a substance and not potential exposure or effects from discarded PVC products As mentioned before, rodents and humans metabolize phthalates differently, and this must be adequately accounted for when comparing the effects in the study. No reference to PVC.
192	I.B.2.i, p. 23 FN 203 – Frederiksen (2007)		This article discusses the metabolism of phthalates in humans. "During the last four to five decades, the male reproductive health in several Western countries seems to have, in fact, deteriorated and the human fertility rates have declined all over the world [6]. The changes include	This article discusses the metabolism of phthalates in humans not potential exposure or effects from discarded PVC products.

			ncreased prevalence of hypospadias, cryptorchidism, testicular cancer [7 – 10] and declining semen quality [11 – 13]." "Phthalates are now widely used all over the world, not only as plasticisers but also as additives in industrial products, including food and personal care products [21, 22, 28]. Especially, the addition of DEHP to polyvinyl chlorides (PVC) for flexibility has been an enormous industrial success [29]. However, due to its toxic and endocrine disrupting effects, DEHP has today been replaced by di-iso-nonyl phthalate (DiNP) as the most commonly used plasticiser in PVC in Europe [30]."	
193	I.B.2.i, p. 23 FN 204 – Latini (2006)	. . . leading experts to conclude that "phthalate exposures could be the leading cause of reproductive disorders in humans."	This article reviews the data that support or discounts the evidence existing to date linking phthalate exposure and the decline of human male fertility, especially in developed countries. "Recent studies have showed that exposure to some phthalates results in profound and irreversible changes in the development of reproductive tract (Foster et al., 2001; Sharpe, 2001) especially in males, raising the possibility that phthalate exposures could be the leading cause of the reproductive disorders in humans. However, it remains controversial whether human male reproductive health is really declining worldwide (Jouannet et al., 2001) and whether endocrine disruptors (e.g. phthalates), perturbing the reproductive process by mimicking or antagonizing steroid hormones action, may play a role in the perceived decline." The study goes on to say that, "[t]he adverse effects of phthalates on male development and reproduction might be mediated from reactive oxygen species (ROS), that have been shown to induce DNA damage and	As mentioned above at line 184, this study singularly focuses on phthalates without considering other endocrine disrupting substances that males are widely exposed to which could contribute to reproductive disorders.

			may accelerate the process of germ cell apoptosis, leading to the decline in sperm counts associated with male infertility."	
194	I.B.2.i, p. 23 FN 205 – Hannas (2013)	Moreover, the analogous "female phthalate syndrome," characterized by various reproductive tract abnormalities and reduced fecundity, is "striking[ly] similar" to the effects of Mayer-Rokitansky-Kuster-Hauser syndrome in women.	The study examines female vulnerability to in utero phthalate exposure and further characterizes a potential model for the human MRKH syndrome. The current study reports female reproductive tract malformations in the Sprague–Dawley rat similar to those characteristics of MRKH syndrome, following in utero exposure to a mixture of 5 PEs. We determined that females are ~2-fold less sensitive to the effects of the 5-PE mixture than males for reproductive tract malformations. Laboratory-grade corn oil (CAS 8001-30-7, Cat# C-8627), and butyl benzyl phthalate (BBP; CAS 85-68-7, Cat# 308501, lot# 08523JQ, purity 98%); dibutyl phthalate (DBP; CAS 84-74-2, Cat# D-2270, lot# 109F0386, purity = 99%); di(2-ethylhexyl) phthalate (DEHP; CAS 117-81-7, Cat# P-6699, lot# 106H3487, purity = 99%); diisobutyl phthalate (DiBP; CAS 84-69-5, Cat# 152641, lot# 103141 C, purity = 99%), and dipentyl phthalate (DPeP; CAS 131-18-0, Cat# 80154, lot# 1151652, purity = 99%) were purchased from Sigma–Aldrich (St. Louis, MO). The doses were delivered in 2.5 µl corn oil per gram body weight. Data were analyzed by one-way analysis of variance (ANOVA) using the general linear measures procedures from the Statistical Analysis Systems (SAS, Inc., Cary, NC). Post hoc comparisons were made using the Least Squared Means procedure on SAS. Data were analyzed as litter means, with the exception of the malformation data which were analyzed both as individual means by Fishers exact test (Sigma Stat, Systat	As discussed in prior comments, the majority of the phthalates cited and relevant to this petition (i.e., DBP, BBP, DiBP) are low molecular weight phthalates that are not commonly used in PVC. DEHP is predominantly used in medical applications and use in PVC has declined in the U.S.

			Software, San Jose, CA) and litter means using ANOVA. Data were considered significant at $p < 0.05$. AGD data were analyzed with and without body weight or the cube root of body weight as a covariate. Results were reported as AGD with no correction for body weight since there was no difference in outcome between the analyses with and without body weight as a covariate in Study 1 or 2 and neither covariate improved the error variance in the analysis. This report highlights the striking similarity of the "female phthalate syndrome" to MRKH syndrome in women, including uterine and vaginal agenesis and renal defects. To date, investigations into the etiology of MRKH syndrome have been unsuccessful in identifying the genetic components of the disease (Morcel et al., 2007; Sultan et al., 2009).	
§ I.B.2.j. Action Taken by Other Governmental Agencies or Regulatory Programs Based on the Health or Environmental Hazard Posed by Discarded PVC, Vinyl Chloride and Phthalate Plasticizers				
195	I.B.2.j, p. 23 FN 206 – Hassan (2004)	Government officials have recognized the problem of marine pollution for nearly a century	*Parenthetical:* (reporting that experts from thirteen maritime powers, including the United States, met in June 1926 to discuss measures to prevent ocean-going vessels from contaminating the marine environment) "Attempts were made (almost seventy-five years ago) to deal with marine pollution issues when, at the invitation of the United States government, experts from thirteen maritime powers (Belgium, Canada, Denmark, France, Germany, Italy, Japan, the Netherlands, Norway, Spain, Sweden, the United Kingdom, and the United States) met in Washington DC in June 1926 to discuss measures to prevent vessel source marine pollution."	Referencing "marine pollution" in general; no direct reference to discarded PVC products. See discussion in main comments.

196	I.B.2.j, p. 23 FN 207 – Harse (2011)	. . . and have implemented a variety of international agreements, federal legislation, and state laws to curb this growing threat.	*Parenthetical*: (reporting international agreements and federal laws pertaining to problem of marine plastic pollution, and observing that "for all of these, plastics continue to reach our oceans") "[T]here is not one, but three separate and distinct international agreements currently in force relating to dumping in oceans. Pursuant to membership in or compliance with these agreements, the U.S. has passed multiple laws to complement and implement these agreements. Yet, for all of these plastics continue to reach our oceans, only to be consolidated within the trash-collecting gyres, including the GPGP. This occurs because the focus of each law is on dumping at sea, even though land-based nonpoint source pollution and runoff during storms are the most significant sources of pollutants, including debris, that are washed into coastal and marine waters.'"	No reference to PVC; discusses marine waste and plastic marine waste in general. As discussed above, PVC is a minor component in MSW, representing less than 3% of plastics estimated to be in MSW.
197	I.B.2.j, p. 23 FN 208 – Barnes (2009)	In the four decades since researchers first reported the presence of plastic litter in the oceans, however, these measures have proven insufficient to prevent the further contamination of the marine environment.	*Parenthetical*: (observing that "the ubiquity and abundance of plastic debris . . . is still growing and even if stopped immediately will persist for centuries") "In the last half-century, there have been many drastic changes on the surface of the planet, but one of the most instantly observable is the ubiquity and abundance of plastic debris. Like many anthropogenic impacts on natural systems, it is one that, despite widespread recognition of the problem, is still growing and even if stopped immediately will persist for centuries." "The accumulation of both macro- and micro-plastics has consistently increased on shores and in sediments for the last four decades (see Thompson et al. 2004; Barnes 2005, respectively)."	As stated previously, CBD's reliance on this article is for general views on plastic waste. There are only two limited mentions of PVC in the reference, which we identified in line 2 above. This includes Table 1 of the reference, which lists PVC at 6% of all plastic discarded as municipal solid waste (MSW) in the U.S. in 2005. Since that time, EPA's SMM Report (see line 169) has tracked the steady decline of PVC in MSW discards to the most recent level reported by EPA in 2020 at approximately 3% of all plastics.

			'Another confounding issue is that the types of plastics present vary between municipal, agricultural and C&D waste. Municipal waste is dominated by containers (e.g. drink bottles) and films (e.g. carrier bags, packaging sheets), agricultural waste may contain large quantities of a single fil and C&D waste may contain polyvinyl chloride (PVC) pipe and large plastic containers. Thus, a municipal stream that contains 10 per cent (by mass) plastics is not equivalent to a C&D stream containing the same percentage."	
198	I.B.2.j, p. 23 FN 209 – Lentz (1987)	For example, although the International Convention for the Prevention of Pollution from Ships, commonly known as MARPOL, explicitly prohibits vessels from dumping plastic waste at sea,	This paper describes and compares the legal regimes that exist to address the presence of plastics and other synthetics in the marine environments. 'Only two of MARPOL's five annexes, (Annexes I and II, governing oil and chemicals in bulk, respectively) have entered into force, having obtained ratification by nations representing at least 50% of the world's shipping tonnage. Annex V of MARPOL contains a provision that prohibits 'the disposal into the sea of all plastics, including but not limited to synthetic ropes, synthetic fishing nets and plastic garbage bags.'"	No reference to PVC; discussing plastic waste generally. This paper is from 1987 and, in 1988, MARPOL Annex V came into force. This practice has been illegal in U.S. waters for decades.
199	I.B.2.j, p. 23 FN 211 – Henderson (2001)		*Parenthetical*: (observing that the amount of plastic debris washing ashore in the Northwestern Hawaiian Islands "show[ed] no sign of diminishing" between 1982 and 1998, "despite implementation of MARPOL Annex V in 1989," and reporting that "Hawaiian monk seals continue to become entangled in marine debris") 'Johnson (1994) conducted beach surveys in Alaska which documented reduced deposition of trawl webbing since implementation of MARPOL V. The amount of entangling debris washing ashore in the NWHI, however, has shown no sign	

			›f diminishing, despite implementation of MARPOL Annex V in 1989. Most of the ›ntangling items we documented (i.e. nets ›nd lines) were clearly from the fishing or ›ther maritime industries. Vessels may be ›ontinuing to dump debris despite MARPOL ›egulations, or lose fishing gear during ›ormal operations." 'Hawaiian monk seals continue to become ›ntangled in marine debris, despite seasonal ›leaning of hazardous items from their ›aulout beaches. Entanglement rates showed ›onsiderable variation during the 17-yr ›bservation period, but rates during 1994-.996 were consistently the highest since ›bservations commenced in 1982."	
200	I.B.2.j, p. 24 FN 212 – NOAA (2008)	Specifically, according to data collected by the National Marine Debris Monitoring Program, as much as 82 percent of shoreline litter may result from activities on land that fall outside the scope of international conventions, including plastics manufacturing and improper waste management.	The report considered persistent solid man-made debris from both land-based and ›cean-based sources and its adverse impacts ›n the marine environment and navigation ›afety. 'The National Marine Debris Monitoring Program (NMDMP), which monitored ›ebris on beaches in the United States, found that land-based sources are ›esponsible for approximately 49 percent of ›arine debris items along beaches, while ›cean-based sources are responsible for ›pproximately 18 percent of debris. The ›emaining shoreline debris, about 33 ›ercent, was identified as general source ›ebris because it could come from either ›and- or ocean-based sources (Sheavly 2007)."	As discussed above at 169, this 2008 study discusses the ›mpact of trash and debris in general; no reference to PVC ›pecifically.
201	I.B.2.j, p. 24 FN 212 – Bean (1987)		*Parenthetical*: (estimating that 90 percent 'of the total pollution entering the oceans . . enters from land-based sources via rivers, ›stuaries and other avenues") 'Each year, some 3-4 billion tons of solid ›aste are produced in the United States, of ›hich an estimated 9 million tons are	This 1987 article is quite dated and, as discussed above, points ›o practices that have been illegal in U.S. waters for decades. The article discusses plastic debris in general; no reference to PVC.

			dumped at sea (National Advisory Committee on Oceans and Atmosphere, 1981). The amount dumped may represent only about a tenth of the total pollution entering the oceans; the majority entering from land-based sources via rivers, estuaries and other avenues."	
202	I.B.2.j, p. 24 FN 213 – Rawlins (2009)	Controls intended to reduce exposure among children also exist in Argentina, Fiji, Japan and Mexico, as well as throughout the European Union. [appears in FN text]	*Parenthetical*: (noting the existence of relevant laws in Austria, Denmark, Finland, France, Germany, Greece, Norway and Sweden) "Bans for phthalates in products intended for children exist in European nations and Japan. Austria, Denmark, Finland, France, Germany, Greece, Norway, and Sweden have all adopted bans. In 2005, the European Parliament made permanent an earlier temporary emergency ban in place since the 1990s. Several other nations, including Argentina, Fiji, Mexico, and Japan have banned the use of phthalates in toys and products intended for use by children."	Discussed children's exposure to phthalates during products intended use and not by exposure from discarded products. Separate legislation and regulation of PVC and phthalates supports the approach advocated in our main comments. *Landfill bans of organic and combustible waste (in Finland and Sweden) and high costs for landfilling were among the main reasons for less PVC going to landfilling and most being incinerated with energy recovery. Denmark is the only country were land- filling of soft PVC is legal, but even here landfilling is minor. The largest share of PVC enters incineration through mixed waste streams. Sometimes this contributes to some technical concerns, such as corrosion of the flue gas cleaning systems in waste- to-energy plants. To prevent chlorine compounds in flue gases, specific legal require- ments are set for minimum incineration temperature (minimum1,000 oC). Lack of do- mestic recycling capacities results in some exemptions from the landfilling bans in Sweden and Finland. In the latter there seems to be a growing political interest in finding better solutions for the management of PVC waste…* *ermany is among the leading countries in PVC waste recycling, reaching 37% of re- cycling for PVC in post-consumers waste. At this recycling level, in 2013 it already achieved one third of the 2020 recycling target for the European PVC industry set by the VinylPlus initiative. The main constituent of separately collected PVC waste is window frames and other PVC profiles. Other PVC waste materials end up in mixed waste flows and go to energy recovery.* *The Netherlands also recycle significant amounts of its PVC waste, which are mainly PVC pipes. Both countries have large PVC manufacturing and recycling com-*

				panies, which are a part of the VinylPlus initiative. Together with other actors within PVC value chains they are organising separate collection of PVC materials.
203	I.B.2.j, p. 24 FN 213 – Center for Health, Environment & Justice (2014)	In addition, Germany, Spain and Sweden have enacted broader bans on the use and disposal of PVC. [appears in FN text]	A factsheet summary of PVC policies across the world. "Sweden first proposed restrictions on PVC use in 1995 and is working towards discontinuing all PVC uses. In Spain, over 60 cities have been declared PVC-free. Germany has banned the disposal of PVC in landfills as of 2005, is minimizing the incineration of PVC, and is encouraging the phase out of PVC products that cannot easily be recycled. Since 1986, at least 274 communities in Germany have enacted restrictions against PVC."	This factsheet no longer appears available and is from an avowed anti-PVC organization. A more authoritative and recent resource is from the Nordic Council of Ministers: *PVC Waste Treatment in the Nordic Countries* available at: [HYPERLINK "https://norden.diva-portal.org/smash/get/diva2:1287469/FULLTEXT01.pdf."]:
204	I.B.2.j, p. 24 FN 214 – Cal. Health & Safety Code § 108935-39	. . . Legislators have enacted similar provisions in California,	*Parenthetical*: (restricting the manufacture, sale and distribution of certain toys and child care articles containing more than 0.1 percent of DEHP, DBP, BBP, DINP, DIDP, or DnOP) Citing California restrictions on the manufacture, sale and distribution of certain toys and childcare articles containing more than 0.1 percent of DEHP, DBP, BBP, DINP, DIDP, or DnOP	These are restrictions on phthalates, not PVC products or discarded PVC products. Separate legislation and regulation of PVC and phthalates supports the approach advocated in our main comments.
205	I.B.2.j, p. 24 FN 215 – Vt. Stat. Ann. Tit. 18, § 1511	. . . Vermont	*Parenthetical*: (restricting the manufacture, sale and distribution of certain toys and child care articles containing more than 0.1 percent of DEHP, DBP, BBP, DINP, DIDP, or DnOP) "(b) Beginning July 1, 2009, no person or entity shall manufacture, sell, or distribute in commerce any toy or child care article intended for use by a child under three years of age if that product contains di-(2-ethylhexyl) phthalate (DEHP), dibutyl phthalate (DBP), or benzyl butyl phthalate (BBP), in concentrations exceeding 0.1 percent.	These are restrictions on phthalates, not PVC products or discarded PVC products. Separate legislation and regulation of PVC and phthalates supports the approach advocated in our main comments.

			(c) Beginning July 1, 2009, no person or entity shall manufacture, sell, or distribute in commerce any toy or child care article intended for use by a child under three years of age if that product can be placed in the child's mouth and contains diisononyl phthalate (DINP), diisodecyl phthalate (DIDP), or di-n-octyl phthalate (DnOP), in concentrations exceeding 0.1 percent."	
206	I.B.2.j, p. 24 FN 216 – Wash. Rev. Code § 70.240.020(1)(c)	. . . and Washington,	*Parenthetical*: (restricting the manufacture, sale and distribution of "children's product[s] or product component[s]" containing, inter alia, "[p]hthalates, individually or in combination, at more than 0.10 percent by weight (one thousand parts per million)") "(1) Beginning July 1, 2009, no manufacturer, wholesaler, or retailer may manufacture, knowingly sell, offer for sale, distribute for sale, or distribute for use in this state a children's product or product component containing the following: (a) Except as provided in subsection (2) of this section, lead at more than .009 percent by weight (ninety parts per million); (b) Cadmium at more than .004 percent by weight (forty parts per million); or (c) Phthalates, individually or in combination, at more than 0.10 percent by weight (one thousand parts per million)."	These are restrictions on phthalates, not PVC products or discarded PVC products. Separate legislation and regulation of PVC and phthalates supports the approach advocated in our main comments.
207	I.B.2.j, p. 24 FN 217 – 40 C.F.R. Part 132	In addition, several state and federal agencies have sought to limit levels of these compounds in the environment	*Parenthetical*: (requiring Great Lakes States and Tribes to adopt, inter alia, provisions sufficient to protect local wildlife from "bioaccumulative chemicals of concern," including six phthalate plasticizers)	No reference to PVC. These are restrictions on phthalates, not PVC products or discarded PVC products. Separate legislation and regulation of PVC and phthalates supports the approach advocated in our main comments.
208	I.B.2.j, p. 24 FN 217 – Cal. Code Regs. Tit. 27, § 25805		*Parenthetical*: (prohibiting businesses from knowingly discharging "chemicals causing reproductive toxicity," including five	No reference to PVC. These are restrictions on phthalates, not PVC products or discarded PVC products.

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			ɔhthalate plasticizers, into any source of ḍrinking water)	ꞩeparate legislation and regulation of PVC and phthalates ꞩupports the approach advocated in our main comments.
209	I.B.2.j, p. 25 FN 218 – Lemos (2011)	However, as this petition describes, the current regulatory scheme is wholly inadequate to prevent the harm likely to result from the continued widespread use and improper disposal of PVC. In addition to ignoring significant sources of human and environmental exposure to phthalate plasticizers, existing laws may suffer from inadequate enforcement. [appears in FN text]	*Parenthetical*: (explaining that federal ɔhthalate restrictions "allow[] states to nfluence policy by adjusting the intensity of ɛnforcement and hence the degree to which nanufacturers are deterred from using ɔhthalates," and observing that an elected ɑttorney general from a "conservative" state night have little incentive to take action in he consumer protection field) 'Although there are questions at the margins ɑbout what constitutes a children's toy, there s a core set of products to which the ban clearly and unequivocally applies. Even in hose circumstances where the federal rule ɔperates unambiguously, enforcement ɑuthority allows states to influence policy ɔy adjusting the intensity of enforcement ɑnd hence the degree to which nanufacturers are deterred from using ɔhthalates. States with a strong commitment o consumer protection can devote resources o identifying and pursuing violations, while hose that wish to court business from toy nanufacturers can abstain from ɛnforcement. Indeed, there is some evidence o suggest that the decision by an elected ɑttorney general to take action in the consumer-protection field is influenced by citizen ideology: Attorneys general from 'liberal" states do more, while those from 'conservative" states do less."	Regulation of toys as products, not discarded products. These are restrictions on phthalates, not PVC products or discarded PVC products. Separate legislation and regulation of PVC and phthalates supports the approach advocated in our main comments.
210	I.B.2.j, p. 25 FN 220 – Wormuth (2006)	Phthalate contamination is now ubiquitous among the population of industrialized nations, and exposure to vinyl chloride "remains a cause for concern."	The study provides a link between the knowledge on emission sources of ɔhthalates and the concentrations of ɔhthalate metabolites found in human urine. 'Recent screening studies in industrialized countries for contaminants in human urine samples have revealed the population's ubiquitous exposure to various plasticizers,	As discussed above at line 166, this is a review of multiple studies using various methods. It discussed exposure to phthalates in daily life, but not as a result of exposure to discarded PVC products. Based on European exposure and data is derived from EU risk assessments/studies.

			he group of phthalic acid diesters (phthalates)."	
211	I.B.2.j, p. 25 FN 220 – Kielhorn (2000)		This article discusses how, from its view, VC remains a cause for concern because of continued exposure and reports of angiosarcoma of the liver. "As a result of the strict occupational exposure limits, no cases of angiosarcoma of the liver (ASL), the tumor associated with VC exposure, have been reported in new workers exposed to VC in those factories since that time. . . But recent epidemiologic, environmental, and biomechanistic findings have opened up new aspects of this chemical. . . VC remains a cause for concern because potential exposure to this chemical and new cases of ASL are still being reported."	Please refer to line 182 above for the most recent U.S. study of ASL cases (Mundt). According to the Mundt study, cases of ASL were limited to workers exposed to high levels of occupational exposure to vinyl chloride up until the regulations were enacted, and that other cancers were insignificant in the study group.
212	I.B.2.j, p. 25 FN 221 – Swan (2005)	According to a 2005 study, one-quarter of U.S. women exhibit concentrations of phthalate metabolites higher than those correlated with irregular sexual development in male infants,	The study presents data to examine AGD and other genital measurements in relation to prenatal phthalate exposure in humans. The study presents "data from the first study to examine AGD and other genital measurements in relation to prenatal phthalate exposure in humans. A standardized measure of AGD was obtained in 134 boys 2–36 months of age. AGD was significantly correlated with penile volume (R = 0.27, p = 0.001) and the proportion of boys with incomplete testicular descent (R = 0.20, p = 0.02). We defined the anogenital index (AGI) as AGD divided by weight at examination [AGI = AGD/weight (mm/kg)] and calculated the age-adjusted AGI by regression analysis. We examined nine phthalate monoester metabolites, measured in prenatal urine samples, as predictors of age-adjusted AGI in regression and categorical analyses that included all participants with prenatal urine samples (n = 85). Urinary concentrations of four phthalate metabolites [monoethyl phthalate	This is a reproductive health study but without a direct link to discarded PVC products. As indicated above, the cited study is "the first study to look at subtle patterns of genital morphology in humans in relation to any prenatal exposure." In addition, the study also includes the following disclaimer: "[o]ur analysis is based on a single measure of AGD, and the reliability of this measurement in humans has not been established."

			MEP), mono-n-butyl phthalate (MBP), nonobenzyl phthalate (MBzP), and nonoisobutyl phthalate (MiBP)] were nversely related to AGI. After adjusting for ige at examination, p-values for regression coefficients ranged from 0.007 to 0.097. Comparing boys with prenatal MBP concentration in the highest quartile with hose in the lowest quartile, the odds ratio for a shorter than expected AGI was 10.2 95% confidence interval, 2.5 to 42.2). The corresponding odds ratios for MEP, MBzP, ind MiBP were 4.7, 3.8, and 9.1, espectively (all p-values < 0.05). We lefined summary phthalate score to quantify oint exposure to these four phthalate netabolites. The age-adjusted AGI lecreased significantly with increasing phthalate score p-value for slope =).009).The associations between male genital development and phthalate exposure seen here are consistent with the phthalate-elated syndrome of incomplete virilization hat has been reported in prenatally exposed odents. The median concentrations of phthalate metabolites that are associated with short AGI and incomplete testicular lescent are below those found in one-quarter of the female population of the Jnited States, based on a nationwide sample. These data support the hypothesis hat prenatal phthalate exposure at environmental levels can adversely affect nale reproductive development in humans."	
213	I.B.2.j, p. 25 FN 222 – Adibi (2003)	. . . and evidence indicates that contamination might be even more prevalent among pregnant women in urban settings.	*Parenthetical*: (reporting that pregnant women in New York City "appear to be exposed [to phthalates] at levels above background levels in the United States, which may have implications for their pregnancy and/or the fetus") '[T]he evidence indicates that pregnant women in New York and Krakow are experiencing a range of exposure levels to	As discussed at line 12, the article mentions the use of DEHP as the 'primary plasticizer' in PVC since the 1930s while introducing the various uses of phthalates in everyday items. As our main comments note, plasticizers and their use has shifted. To that end, current NHANES metabolite data shows exposure to phthalates has declined significantly in the U.S. BA Beckingham, *et al*., *Phthalate exposure among U.S. college-aged women: Biomonitoring in an undergraduate*

			ɔhthalates with some extreme values that nay be associated with a biological esponse. The New York women appear to ɔe exposed at levels above background evels in the United States, which may have mplications for their pregnancy and/or the etus. These results require further nvestigation. A molecular epidemiological study is being carried out to more horoughly characterize exposures in these wo cohorts and to incorporate placental narkers of in utero endocrine disruption that nay be related to placental function and ɔregnancy outcomes."	*student cohort (2016-2017) and trends from the National Health and Examination Survey (NHANES, 2005-2016)*, PLoS ONE 17(2): e0263578 (2022), https://doi.org/10.1371/journal.pone.0263578.
§ I.B.2.k. Other Appropriate Factors				
214	I.B.2.k, p. 25 FN 223 – Hamlin (2011)	Moreover, recent research indicates that exposure pathways "outside the scope of traditional toxicity testing" might result in additional harm.	'Assessing whether environmental concentrations are high enough to warrant concern has been traditionally based on whether environmental concentrations are below 'no observable effect levels' (NOAL) determined by traditional toxicity testing in the laboratory. However, it is becoming clear that exposure scenarios outside of the scope of traditional toxicity testing, such as low level exposures during critical developmental windows, or exposure to multiple chemicals at a time, can dramatically alter the developmental trajectory of an embryo. Although it is often difficult to assign causality in instances of developmental dysfunction in wildlife due to the complexity of the exposure environment, there is nonetheless a considerable body of literature linking exposure of wildlife living in contaminated environments with a variety of reproductive and developmental disorders (Colborn and Clement, 1992; Edwards et al., 2006c; Milnes et al., 2006; Tyler et al., 1998)."	No reference to PVC products; focus is on exposure to phthalates and whether NOAL is the correct criteria.

215	I.B.2.k, p. 25 FN 224 - *Id.*	For example, low doses of phthalates and other endocrine-disrupting chemicals often produce health effects different from or more severe than those associated with higher concentrations.	"Also overlooked in traditional toxicity testing are nonmonotonic dose responses, whereby very low chemical exposures cause deleterious effects that are either less severe or not seen at all at higher doses. Traditional toxicity testing shows that using high doses of estrogen during embryonic development of male mice inhibits prostate development (vomSaal et al., 1997). However, very low level exposure to estrogen or estrogenic compounds causes a significant increase in fetal prostatic glands. This leads to a twofold increase in prostatic ARs in adulthood and a concomitant increase in prostate size (vomSaal et al., 1997). Physiological responses caused by low doses should be distinguished from toxicological responses caused from unnaturally high exposures (Fig. 3). Accordingly, traditional toxicity testing using elevated doses should not be the sole factor in determining thresholds of safety."	
216	I.B.2.k, p. 25 FN 226 – Mankidy (2013)	Simultaneous exposure to multiple phthalates or to a single phthalate mixed with other environmental pollutants, might elicit a synergistic response.	This study investigated cytotoxicity, endocrine disruption, effects mediated via AhR, lipid peroxidation and effects on expression of enzymes of xenobiotic metabolism caused by di-(2-ethy hexyl) phthalate (DEHP), diethyl phthalate (DEP), dibutyl phthalate (DBP) and benzyl butyl phthalate (BBP) in developing fish embryos. "Risk assessment with individual phthalates have concluded that exposure to receptors is often several orders of magnitude below the toxicological threshold (Kamrin, 2009). However, in reality a receptor is exposed to multiple phthalates simultaneously. The fact that multiple phthalates can act through the same biological pathway in vivo further compounds their impact on the organism and highlights the importance of investigating the effects of exposure to mixtures of phthalates (Howdeshell et al., 2008). Lastly, the heterogeneity of response	This is a toxicology study regarding potential effects of exposure to plasticizers not potential exposure or effects from discarded PVC products. As explained in numerous comments above, CBD is conflating the use of low MW phthalates like DEP, DBP, and BBP with PVC applications, when they are actually widely used in non-PVC materials that provide far greater exposures to individuals than what could be expected from any PVC materials.

			to phthalates in the study adds another layer of complexity demonstrating the need to investigate multiple endpoints simultaneously while assessing human health and ecological health." "MVLN cells were propagated in DMEM/F-12 media containing 10% FBS at 37 ◦C, 5% CO2. Cytotoxicities of phthalates were determined by exposing 8 × 104 MVLN cells to DEHP, DEP, DBP or BBP (Sigma–Adlrich, St. Louis, MO) for a period of 24 hr. WST-1 reagent (Roche Applied Science, Indianapolis, IN) was used to determine metabolically active cells at the end of the incubation period according to the manufacturer's recommendation."	
217	I.B.2.k, p. 25 FN 226 – Hamlin (2011)		"This study shows that for certain harmful effects, such as hypospadias, the mixture of anti-androgens used in this study caused a worse response than would have been predicted from their individual toxicities. This type of synergistic response makes it difficult to estimate the outcome of mixture exposures, as traditional toxicity testing would result in an underestimate of the exposure risks."	No reference or correlation to PVC or discarded PVC products.
218	I.B.2.k, p. 25 FN 226 – Howdeshell (2008)		The article summarizes research on the cumulative effects of binary and complex mixtures of phthalates with each other and/or with other androgen-disruptive chemicals on male reproductive development and how laboratory animal research regarding the effects of phthalate esters on reproductive development furthers our understanding of the potential health risks of such chemicals on the developing human. The article concludes that: "Continued research efforts are needed to assess the cumulative risk of chemical mixtures on human reproductive health. Our research in laboratory rats has demonstrated that	The article does not establish a connection between discarded PVC products and phthalate exposure.

			mixtures of phthalate esters with one another, and with other anti-androgenic chemicals, can alter reproductive development and hormone production in a largely dose-additive fashion.”	
219	I.B.2.k, p. 25 FN 226 – Jobling (1995)		*Parenthetical*: (noting that scientific literature suggests that “measuring the total estrogenic burden due to environmental contaminants may have more relevance than assessing exposure by measuring levels of individual estrogens alone,” because “environmental estrogens may act cumulatively”) “[T]he effects of simultaneous exposure to a variety of estrogenic chemicals should be investigated. Since all of the estrogenic chemicals discovered to date are lipophilic, they probably co-exist in fate and body fluids of exposed individuals. Much of the current literature suggests that environmental estrogens may act cumulatively and that measuring the total estrogenic burden due to environmental contaminants may have more relevance than assessing exposure by measuring levels of individual estrogens alone.”	This reference discusses alternative methods of assessing estrogenic burden, not potential exposure or effects from discarded PVC products. However, the point referenced in the parenthetical undercuts CBD’s argument to regulate phthalate plasticizers as a class and much of the other literature cited, which focused on exposure.
220	I.B.2.k, p. 25 FN 228 – Frederiksen (2007)	In addition, the recent discovery of additional phthalate metabolites indicates that human exposure probably exceeds previously published estimates.	Discusses the metabolism of phthalates in humans. “Furthermore, previously published exposure estimates based on excretion in urine are probably underestimated. In particular, the estimates of the exposure to the long-branched phthalates (DEHP and DiNP) based on urine excretion of their primary metabolites MEHP and MiNP might result in too low estimates, because the mono- ester phthalates are, in fact, not the major excreted metabolites of these compound. As mentioned, secondary metabolites of the long-branched phthalates have been identified and characterised during the last years [37, 62, 68, 69, 92], and	Discusses the metabolism of phthalates in humans, not potential exposure or effects from discarded PVC products. In contrast to CBD’s assertion, current NHANES metabolite data shows exposure to phthalates has declined significantly in the U.S. BA Beckingham, *et al.*, *Phthalate exposure among U.S. college-aged women: Biomonitoring in an undergraduate student cohort (2016-2017) and trends from the National Health and Examination Survey (NHANES, 2005-2016)*, PLoS ONE 17(2): e0263578 (2022), https://doi.org/10.1371/journal.pone.0263578.

			co-measurement of these will in the future give more precise estimates of human exposures to phthalates.”	
221	B.2.k, p. 25 FN 229 – Stern (2008)	Because the majority of chemical compounds used in PVC production remain untested, existing toxicity data likely underestimate risks arising from the improper disposal of discarded PVC.	This review evaluates the literature on the occurrence of regulated and unregulated substances in drinking water related to the use of plastic pipes, characterizes potential health hazards, and describes uncertainties associated with human health and exposure in need of further research. “Migration may vary significantly among pipes, depending on the materials/resins used in manufacturing and extrusion, differing location/environmental conditions and usage patterns. The possible health effects of some leachates have been studied in animals and/or humans. The majority of constituents or additives in plastic pipes have not been monitored and/or evaluated toxicologically. Chemical compounds are not routinely measured at the tap and the potential for additive or synergistic adverse health effects in situ is not well characterized.”	This study discusses intended product use, not the management of discarded PVC products. The claim that ‘the majority of chemical compounds in PVC product remain untested” is false and misleading. As the study notes, very little data on leachates from PVC and CPVC pipe is available because the pipe and fitting materials are tested and certified to meet NSF Standard 61, which was developed in 1976 and adopted by EPA in 1986 for assuring the health and safety of drinking water components.